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The Croonian Lectures
ON
THE NATURAL HISTORY AND PATHOLOGY OF PNEUMONIA.

BY THE LATE J. W. WASHBOURN, C.M.G., M.D., F.R.C.P. LOND.,

CONSULTING PHYSICIAN TO H.M. FORCES IN SOUTH AFRICA; PHYSICIAN TO GUY'S HOSPITAL AND THE LONDON FEVER HOSPITAL, &C.

Delivered before the Royal College of Physicians of London on Nov. 11th, 13th, 18th, and 20th, 1902, by W. HALE WHITE, M.D., F.R.C.P. Lond.; *and edited by* J. W. H. EYRE, M.D. Durh.

LECTURE III.[1]

Delivered on Nov. 18th.

Bactericidal action of various sera.—In 1890 Baring and Nissen showed that the blood serum of a normal rabbit possessed no bactericidal action, the method which they adopted being that of plate cultivations. They found that the serum of a normal guinea-pig as a rule had no bactericidal property, but the serum of one guinea-pig which they examined possessed marked bactericidal properties. Kruse and Pansini in 1892 repeated these experiments; they also found that the normal rabbit serum possessed no bactericidal action, and they found the same result with the normal guinea-pig serum as did Baring and Nissen. The serum of the dog and human ascitic fluid gave similar results to those of the guinea-pig. The serum of a sheep had marked bactericidal properties, and so too had the serum of immunised rabbits. Eyre and I made a number of observations upon this subject.[2] We used normal rabbit serum, normal fowl serum, normal guinea-pig serum, the serum of an immunised rabbit, and the serum of an immunised pony. We obtained exactly similar results to the other observers in reference to the normal rabbit serum and to the normal guinea-pig serum; the normal fowl serum never possessed any bactericidal properties; the serum of the immunised rabbit and of the immunised pony in some cases possessed definite bactericidal properties and in other cases possessed none.

TABLE IV.—*Normal Rabbit's Serum.*

—	Age of serum in hours.	Number of colonies at 0 hours.	2 hours.	4 hours.	12 hours.	24 hours.	36 hours.	48 hours.	60 hours.
A	0	24	243	515	∞	∞	∞	∞	2300
B	300	181	1360	1914	∞	∞	1	—	—

The serum used in the Experiments A and B was obtained from the blood of the same rabbit.

TABLE V.—*Normal Fowl's Serum.*

—	Age of serum in hours.	Number of colonies at 0 hours.	4½ hours.	12 hours.	18 hours.	24 hours	48 hours.	72 hours.
A	0	3	8	1	—	∞	500	—
B	12	4	1	∞	∞	2000	—	—
C	12	38	65	∞	—	1348	—	—
D	18	747	89	∞	—	∞	0	—
E	36	669	∞	∞	—	315	—	—
F	36	11	8	63	—	∞	12	0
G	396	59	81	∞	—	67	0	—

Experiments A and D were made with the serum from one fowl, E, B, and F from a second, and C and G from a third.

TABLE VI.—*Normal Guinea pig's Serum.*

—	Age of serum in hours.	Number of colonies at 0 hours.	5 hours.	12 hours.	24 hours.	40 hours.	48 hours.	72 hours.
A	0	541	3100	∞	∞	0	—	—
B	0	151	2543	∞	∞	144	729	0
C	12	480	854	3352	∞	—	—	—
D	142	34	129	∞	∞	—	8	—
E	180	224	170	180	0	—	—	—

Experiment A was made with the serum from one guinea-pig, B and D from a second, C from a third, and E from a fourth.

TABLE VII.—*Serum of Immunised Pony.*

—	Age of serum.	Number of colonies at 0 hours.	4 hours.	12 hours.	24 hours.	36 hours.	48 hours.
A	8 weeks.	122	51	40	∞	∞	—
B	9 weeks.	594	369	310	75	6	0

Experiments A and B were made with the same serum, but at different ages.

TABLE VIII.—*Serum of Immunised Rabbit.*

—	Age of serum in hours.	Number of colonies at 0 hours.	4 hours.	12 hours.	24 hours.	48 hours.
A	24	97	130	∞	∞	969
B	48	29	36	∞	∞	1364
C	48	287	104	105	54	0

Experiments A, B, and C were all made with the serum of the same rabbit, removed at different times.

Pane in his experiments made the following observations. He found that the serum of an immunised animal acted most powerfully when it was mixed with a cultivation and the mixture injected. The serum of an immunised cow and of an immunised donkey, on the other hand, acted more powerfully when the serum was injected directly into the circulation and the cultivation injected under the skin. His explanation is that the immunity produced by the serum was an active immunity and that the rabbit serum produced its effect more rapidly than the serum of the other animals. It is quite possible, however, that the difference may be due to the presence of a specific addiment in the rabbit serum which is absent in other sera, a question which we shall discuss later. Mennes[3] found that the serum of immunised rabbits by itself exercised no bactericidal influence. If, however, to this serum there were added normal white blood corpuscles or the white blood corpuscles of an immunised animal the growth was hindered. He also found that when leucocytes were added to normal serum to which pneumococci were added they did not inglobe the cocci, but if in place of normal serum immune serum was used the leucocytes were capable of inglobing the cocci; the immune serum so acted upon the cocci as to render them sensitive to the action of the leucocytes.

Method of recognising pneumococci in lesions.—The microscopical examination of sputum is not satisfactory for determining the presence of pneumonia; Gamaleia states that out of a number of observations the two cases in which the largest number of pneumococci were found in the sputum were not cases of pneumonia. Some years ago I made a number of observations with reference to this point and I found that it was exceedingly difficult to detect pneumonia by a microscopical examination of the sputum. Lindsay (M.D. thesis, Cambridge, 1902) has recently made an extensive series of observations in this connexion with similar results. The method of inoculation appears to be the only certain method of finding the cocci, but when we are dealing with sputum we have the difficulty that pneumococci are frequently normally present in the mouth. Honl[4] states that by

[1] Lectures I. and II. were published in THE LANCET of Nov. 15th (p. 1301) and 22nd (p. 1378), 1902, respectively.

[2] Further Researches upon the Pneumococcus, Journal of Pathology and Bacteriology, January, 1898.

[3] Zeitschrift für Hygiene und Infektions-Krankheiten, Band xxv., Heft 3, p. 413.

[4] Centralblatt für Bakteriologie, Band xxiii., p. 274.

injecting pneumococcic sputum under the skin of the ear of a rabbit a marked œdema occurs in 24 hours; the swelling consists of leucocytes, fibrin, and cocci. The sputum from other diseases does not produce the same effect, inasmuch as the mouth pneumococci are either present in too small numbers or are not sufficiently virulent. Gamaleia examined by the method of inoculation the lungs in 12 cases of pneumonia and in each case he demonstrated the presence of the pneumococcus. He says that mice are more susceptible than rabbits to inoculation and he attributes Netter's failure to find pneumococci in 25 per cent. of his cases to the use of rabbits instead of mice. He also says that Weichselbaum's failure to find pneumococci in 35 out of 129 cases was due to his failure to use cultural methods in addition of inoculation.

Pneumococci in nature.—Netter [5] found pneumococci in the dust from hospital wards by injecting guinea-pigs; in two animals pneumococci were found after death. Emmerich also isolated pneumococci from the dust of a ward, and Uffelmann in the air of a cellar. Maximovitsch [6] found pneumococci in the dust of a hospital. Eyre and I examined dust obtained from various sources by means of inoculation experiments upon mice and were able to demonstrate the presence of the pneumococcus in dust obtained from a hospital ward (Mary Ward, Guy's Hospital) and also from a cupboard in a physiological laboratory (Guy's). Inoculations of specimens of dust from the street, the entrance-hall of a hospital (Charing Cross), and also some of its wards gave negative results. Pneumococci were frequently found in the mouths of healthy individuals: Netter found them in 15 per cent. of the saliva of healthy persons, Sternberg in 20 per cent. They were originally first found by Pasteur in the saliva of patients suffering from hydrophobia. Eyre and I examined the apparently normal saliva from 13 perfectly healthy individuals and from seven hospital patients suffering from such diseases as occupation paralysis, chlorosis, &c.—20 persons in all. By injecting the saliva into the subcutaneous tissues of the rabbit we isolated the pneumococcus from the saliva of six of these—i.e., 30 per cent. It is commonly found in the nose of healthy individuals. Netter [7] and Kurth found it in the nose. Von Besser [8] found it in 14 out of 57 individuals. Arnold Lawson found the pneumococcus in two out of 200 healthy conjunctival sacs, while Gasperini found it in eight out of 10 cases.

Of still greater importance is the presence of pneumococci in the lungs of healthy individuals. Dürck [9] examined the lungs of 13 children who had died from some other disease; he found pneumococci 12 times, in most cases mixed with other bacteria; in the thirteenth case other bacteria were found, but not the pneumococcus. He also examined the lungs of dogs and horses, and out of 15 cases he found pathogenic bacteria, only one being sterile; among these bacteria were pneumococci. Boni [10] found that the lungs of guinea-pigs were almost always sterile, but in certain cases pneumococci were present. Dogs almost always have bacteria in their lungs; in 20 cases only six were sterile; the remaining 14 contained bacteria, and of these five contained pneumococci.

Other localisations of the pneumococcus.—Pneumococci are found not only in the affected lung but also frequently in other parts of the body. Nazari [11] found cocci in the blood during life in four out of 17 cases; he also examined the spleen, but only found pneumococci present there when they were present also in the blood. F. W. White of Boston examined 10 cases of pneumonia during life; in three of these pneumococci were present and they were all fatal, and the seven other cases proved fatal although no pneumococci were found. Kohn [12] examined 32 cases; in nine of these cases pneumococci were found, seven of which were fatal; in the remaining 23 no pneumococci were found, and of these five were fatal. Banti [13] says that cocci are frequently present in the blood in pneumonia, and indeed believes that they are probably always present. Pernice and Alessi [14] found pneumococci in the blood and various organs in two cases of fatal pneumonia. We can thus readily understand how it is that with pneumonia metastatic lesions are frequently found. Again, the pneumococcus may actually enter through the lungs and produce metastatic lesions without leaving behind any trace of inflammation in the lung itself. This, indeed, we know experimentally, as the following experiment performed by Eyre and myself shows. A rabbit was anæsthetised and a catheter passed through the larynx into the trachea. A cubic centimetre of a broth cultivation of the pneumococcus was poured down the catheter. The animal was killed 15 days later. There were marked pleurisy and pericarditis, but the lungs were quite normal. The pneumococcus may also enter the body in other ways than through the lung, and we consequently find various lesions caused by the pneumococcus and not preceded by pneumonia, such as septicæmia, pleurisy, endocarditis, arthritis, meningitis and cerebritis, nephritis, parotitis, peritonitis, pericarditis, colitis and gastritis, cellulitis, osteomyelitis, acute otitis media, and conjunctivitis.

As it is not my purpose to enter fully into these various lesions a brief mention of some of the more important and interesting will suffice. I have already mentioned that pneumonia is frequently accompanied by septicæmia—that is to say, that the cocci escape from the lungs and multiply in the blood and other tissues. J. Pelnar [15] quotes a case of pneumococcic septicæmia described by Hlava. He also describes two cases of his own, one a child three years old who died from septicæmia and meningitis; pneumococci were found in the meningeal pus and in the spleen. The other was a case of puerperal septicæmia in which pneumococci were found in the pus from the tonsils, in the tracheal mucus, in the spleen, and on the surface of the brain. Foulerton [16] also describes a case of generalised septicæmia.

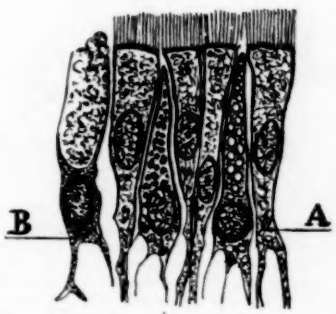
The pleurisy found in pneumonia is always due to the pneumococcus; so, too, are the empyemata following this disease. Many cases of primary empyema and pleurisy are also due to the pneumococcus. I described a fatal case of acute empyema in an adult which was caused by the pneumococcus, and another case in a child in whom both lungs were covered with thick tenacious fibrin with a slight turbid exudation, due to the same micro-organism.[17] Infective endocarditis following upon pneumonia has been described by many observers, Weichselbaum [18] being one of the first. Harbitz records three cases of infective endocarditis due to the pneumococcus, Banti eight, and Pakes one. A number of cases of arthritis have been described during the course of pneumonia. Vogelius [19] describes two cases; Macaigne and Chipault [20] also described two cases; Cave [21] two cases; and seven cases out of 817 by Nathan Raw.[22]

A number of cases of meningitis following on pneumonia have been described by Weichselbaum, Netter, Ortman, and Zoerkendorffer. In some cases the meningitis is primary. Henke described four cases [23]; Wolff [24] collected a number of cases of meningitis from the literature and found that pneumococci were the cause in 42·2 per cent.; Lazarus-Barlow [25] described two cases of posterior basal meningitis due to the pneumococcus; Dr. W. H. Brodie, Dr. W. G. Rogers, and Dr. E. T. E. Hamilton [26] describe an epidemic form of cerebro-spinal meningitis consecutive to rhinitis and resulting from the invasion of the tissues by the pneumococcus, which occasionally occurs amongst the Kaffirs working in the mines of the Rand. Marchaux [27] states that pneumonia is common amongst the natives and that it is not infrequently complicated with cerebro-spinal meningitis; in some cases the meningitis occurs without pneumonia. These latter he believes are due to extension of inflammation from the frontal sinus; in all the cases examined pneumococci were present. He believes that the "sleeping sickness" is due to the effects of chronic pneumococcal meningitis. Dr. George Batten at the Pathological Section of the British Medical Association, 1901, mentioned a case of abscess of the brain following upon pneumonia.

[5] La Semaine Médicale, 1897, p. 209.
[6] Centralblatt fur Bakteriologie, Band xviii., 1895, p. 128.
[7] Archives de Médecine Expérimentale, 1890, p. 687.
[8] Ziegler's Beiträge, 1899, Band vi.
[9] Deutsches Archiv fur Klinische Medicin, Band lviii., 1897, p. 368.
[10] Ibid., Band xlix., 1901, Heft 506.
[11] La Riforma Medica, 1897, No. 96.
[12] Deutsche Medicinische Wochenschrift, 1897, No. 9.
[13] Lo Sperimentale, vol. xliv., 1890, p. 349.
[14] La Riforma Medica, vol. vi., 1890, pp. 662-668.
[15] Wiener Klinische Rundschau, 1899, p. 707.
[16] Transactions of the Pathological Society of London, January, 1902.
[17] Transactions of the Royal Medical and Chirurgical Society, vol. lxxvii., 1894.
[18] Wiener Medicinische Wochenschrift, Band lxxxviii., p. 35.
[19] Hospitalstidende, 1897, p. 281.
[20] Revue de Médecine, 1891, No. 9.
[21] THE LANCET, Jan. 12th, 1901, p. 82.
[22] Brit. Med. Jour., Dec. 12th, 1901.
[23] Arbeiten aus dem Pathologischen Institut, Tubingen, Band ii., 1896, Heft 2.
[24] Berliner Klinische Wochenschrift, 1897, No. 10.
[25] Pathological Section, British Medical Association, 1901.
[26] THE LANCET, Oct. 22nd, 1898, p. 1045.
[27] Annales de l'Institut Pasteur, tome xiii., p. 193.

Faulhabe [28] examined 35 cases of pneumococcal infection, chiefly croupous pneumonia, and found cocci in the kidneys, sometimes in large numbers; they produced parenchymatous changes and small foci of leucocytes. Fränkel and Reiche found pneumococci in the kidneys in 22 out of 24 cases examined post mortem. Tuffier [29] describes a case of peri-nephritis due to the pneumococcus following upon broncho-pneumonia. Duplay [30] describes a case of parotitis following pneumonia; another case is mentioned. Testo [31] describes another case. Marchaux [32] states that pneumonia amongst the natives is sometimes complicated with vaginitis and peritonitis. Sévestre [33] describes a case of primary peritonitis occurring in a child, eight years old, due to the pneumococcus. Dieulafoy quotes 17 cases, chiefly in children. Menefier and Legraux also describe two cases. Bryant [34] also describes two cases. Pericarditis is not an infrequent complication of pneumonia and is caused by direct extension of the cocci. Still (British Medical Association) describes cases in children which appear to have been primary. Foulerton describes a case of pneumococcal gastritis which gave rise to a generalised septicæmia. [35] Foulerton [36] also mentions a case of pneumococcal cellulitis and another of circumscribed abscess of the finger. Fischer and Levy described two cases of acute osteomyelitis due to the pneumococcus. Many cases of acute otitis media have been described by Zaufal, [37] Netter, Weichselbaum, and Bordoni-Uffreduzzi; Green [38] found pneumococci present in 10 out of 73 cases examined; he also found pneumococci in 23 out of 144 cases of acute mastoiditis. Many observers have found pneumococci in acute conjunctivitis. Gifford [39] found pneumococci by microscopical

FIG. 7.

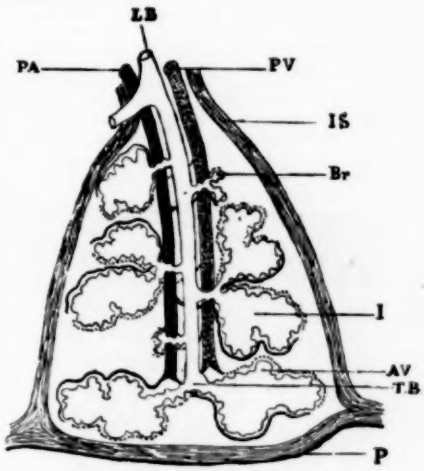


Ciliated epithelium of trachea, after Schäfer. A, Ciliated epithelium cell. B, Mucus-secreting cell.

examination in 39 out of 40 cases of conjunctivitis, and in 12 of these by means of cultivations. Cuénod [40] also describes cases. Groenouw [41] found pneumococci in five out of 100 cases of conjunctivitis in newly-born children. Gonin [42] found pneumococci in 11 out of 365 cases of conjunctivitis. Gasparini [43] found pneumococci in six cases of keratohypopion, also in two out of four cases of panophthalmitis.

The anatomy of the lung.—Before proceeding to detail my observations on the fate of particulate matter, whether living (e.g., bacteria) or dead (e.g., particles of dust, &c.), introduced into the lung it will be well to summarise briefly the structure and arrangement of the various tissues which go to form the organs of respiration. The respiratory tract starts at the nose and mouth, passes through the pharynx to the larynx, and thence along the trachea to the bronchi, which enter the lung at its root. The entrance of the nostrils is guarded by hairs, and the mucous membrane of the nose and that of the upper part of the pharynx are lined with ciliated epithelium. The buccal mucous membrane and the lower part of the pharynx are lined with stratified epithelium. The greater part of the larynx and the trachea and bronchi are lined with ciliated epithelium (Fig. 7). The whole of the mucous membrane is plentifully supplied with mucous cells and with mucous glands, so that its surface is covered with mucus. There is, in addition, abundant lymphoid tissue which in places, as, for instance, at the tonsils, comes close to the surface and from which are derived leucocytes which are found free on the surface of the mucous membrane. The whole of this arrangement is adapted to free the air which enters the lungs from solid particles, whether bacteria or otherwise. Such particles are entangled in the mucus and are conveyed by the ciliated epithelium to the orifices of the nose and mouth, whence they are discharged. Some of the particles are also taken up by the leucocytes and are similarly discharged. I shall describe this process in detailing the results of inhalation experiments. The lung itself is essentially a compound racemose gland, modified to serve the respiratory functions, the bronchi representing the ducts and the alveoli the acini of the gland. Each bronchus divides dichotomously, and the resulting branches continue to divide in a similar manner

FIG. 8.



Scheme of lobule and lung, after Landois and Stirling. P. A., Pulmonary artery. P. V., Pulmonary vein. T. B., Terminal bronchus. Br., Bronchiole. I., Infundibulum. A. V., Air vesicles. I. S., Interlobular septum. P., Pleura. L. B., Lobular bronchus.

into smaller and smaller branches. From these branches others are given off at right angles, and these again divide and ultimately end, when they reach the size of 0·1 to 0·16 millimetre, as alveolar passages or bronchioles, each opening into the apex of a pyriform sacculus, the infundibulum (Fig. 8). The wall of the infundibulum is made up of a number of cup-shaped sacculi, the air cells or alveoli, many of which are not simple but compound, having similar alveoli opening into them (Fig. 9). The wall of the alveolar passage which opens into the apex of the infundibulum is similarly sacculated with alveoli but not to the same extent. The alveoli of adjacent infundibula are closely packed together and only separated by delicate connective tissue. The infundibula with their alveoli correspond to the smallest lobule of a racemose gland, and the alveolar passages to the commencement of the duct.

What is usually described as a lobule of the lung consists of a group of these infundibula and alveolar passages united together by delicate connective tissue and separated by thicker bands of connective tissue from adjacent groups. A lobule measures from 0·3 centimetre to three centimetres in diameter and can be seen with the naked eye in the adult

[28] Ziegler's Beiträge, Band x., 1891.
[29] Le Bulletin Médical, 1892, p. 865.
[30] La Semaine Médicale, 1891, No. 2.
[31] International Congress of Medicine in Rome, 1899.
[32] Annales de l'Institut Pasteur, tome xiii., p. 193.
[33] Bulletin et Mémoires de la Société Médicale des Hôpitaux de Paris, 1890, No. 17.
[34] Pathological Section of the British Medical Association, 1901.
[35] Transactions of the Pathological Society of London, January, 1902.
[36] Ibid.
[37] Prager Medicinische Wochenschrift, 1888-89.
[38] Journal of the Boston Medical Society, vol. iii., p. 93.
[39] Archiv für Augenheilkunde, Band xxxiv., p. 134.
[40] La Semaine Méticale, 1896, p. 226.
[41] Von Graefe's Archiv für Ophthalmologie, Band iii., p. 1.
[42] Revue Médicale de la Suisse Romande, 1899, Nos. 2 and 3.
[43] Eleventh International Congress of Medicine in Rome.

lung, especially on the surface, where the bands of connective tissue separating adjacent lobules are usually pigmented. In the child's lung the separate infundibula, which vary from 0·25 millimetre to one millimetre in size, can also be seen, inasmuch as the connective tissue between adjacent

FIG. 9

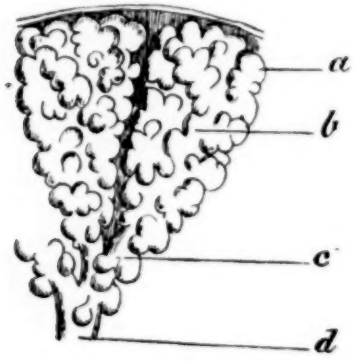
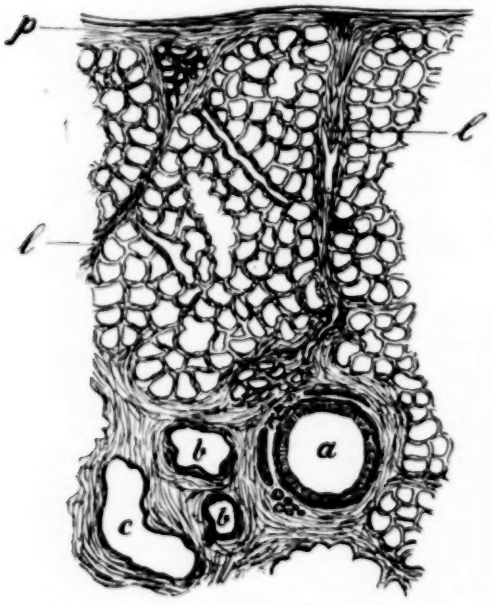


Diagram showing two infundibula. *a*, Alveoli. *b*, Infundibulum. *c*, Alveolar passage. *d*, Terminal bronchus.

infundibula is more developed than in the adult (Fig. 10). The alveoli are cup-shaped and rounded or polygonal in section, according as the lung is collapsed or inflated. They vary in size from 0·05 to 0·1 millimetre in diameter in the adult and 0·03 to 0·06 millimetre in the child. The structure of these various parts is best understood by

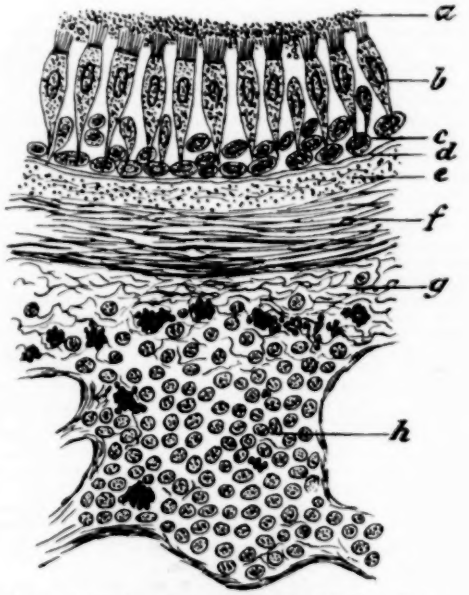
FIG. 10.



Section of lung, showing lobule and pleura, after Landois and Stirling. *a*, Small bronchus. *b*, Pulmonary artery. *c*, Pulmonary vein. *l*, Interlobular septum. *p*, Pleura.

starting with the bronchi. (Fig. 11.) A small intrapulmonary bronchus is formed of a mucous membrane consisting of a stratified columnar ciliated epithelium resting on a basement membrane, outside which is a layer of connective tissue with elastic fibres and still further out a layer of unstriped muscular fibres. Covering the mucous membrane is a fibrous coat consisting of connective tissue containing cartilage, mucous glands, and adenoid tissue. As the bronchi decrease in size their walls become reduced; the cartilage and glands are the first to disappear and are not present in tubes less than 0·5 milli-

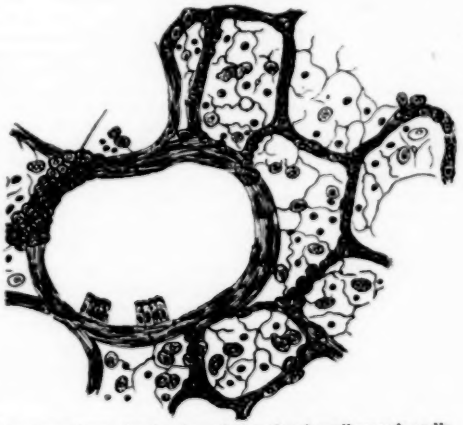
FIG. 11.



Section of bronchus, after Landois and Stirling. *a*, Precipitated mucus. *b* Ciliated epithelium. *c*, Debove's membrane. *d*, Elastic basement membrane. *e*, Inner fibrous layer. *f*, Muscle layer. *g*, Outer fibrous layer. *h*, Adenoid tissue and pigment.

metre. The muscular fibres exist longer and are still seen as a thin layer in the alveolar passages. The epithelium in the alveolar passages has become reduced to a single layer of low polyhedral non-ciliated cells. In the alveoli further changes occur, the walls consisting only of a thin outer coat

FIG. 12.



Section of lung, showing large flattened and small granular cells.

of almost homogeneous connective tissue with elastic fibres and capillaries derived from the pulmonary artery, lined with epithelium. The epithelium consists of flattened epithelial cells, often without nuclei, and small polygonal granular nucleated cells, lying in the interstices of the capillary loops (Fig. 12)

The various structures of the lung are bound together by a delicate framework of connective tissue, which is very thin between the alveoli; and which forms thicker bands between the lobules and a sheath surrounding the bronchi and their divisions, accompanying the blood-vessels. The connective tissue is continuous at the surface of the lung with the connective tissue of the pleura and at the root of the lung with the mediastinal connective tissue. The whole of the lung is thus divided up by a framework of connective tissue into lobes and lobules. The connective tissue forms a thin layer under the pleura which joins at the root of the lung, the connective tissue accompanying and forming a sheath to the bronchus, pulmonary artery and vein and their divisions through the lung. At the root of the lung the connective tissue is continuous with that in the posterior mediastinum and thus with the connective tissue under the parietal pleura.

The blood-supply of the lung.—The lung receives its blood-supply from two sources—from the pulmonary artery and from the bronchial arteries. The pulmonary arteries and veins accompany the bronchial tubes, but branch more frequently (see Fig. 8). The arteries break up into capillaries which are found in the alveolar walls and are separated from the external air only by the alveolar epithelium. The pulmonary veins form frequent anastomoses. The smallest bronchioles are supplied both by pulmonary and bronchial arteries. The bronchial arteries follow the bronchial tubes and supply the bronchial glands, the walls of the larger blood-vessels, the bronchial tubes, and the interlobular connective tissue, and form a plexus under the pleura. The blood is returned chiefly by the bronchial veins, which open into the azygos veins, and partly by the pulmonary veins. The arrangement of the lymphatics is of considerable importance in regard to the entrance and spread of infection through the lung. The lymphatics of the lung begin as small canaliculi and lacunæ in the alveolar walls between the capillaries. They are separated from the air in the alveoli by the alveolar epithelium and they empty into the perivascular lymphatics and communicate with the lymphatics in the interlobular septa. In the walls of the bronchi and their divisions there are abundant similar lymph canaliculi and lacunæ which open into the peribronchial lymphatics. The peribronchial and perivascular lymphatics freely anastomose and end by entering the bronchial glands. Adenoid tissue is found in connexion with the lymphatics in the bronchial walls, around the alveolar passages, and in the inter-alveolar and interlobular connective tissue. There is a rich plexus of lymphatics in the pleura, into which the lymphatics of the superficial alveoli open. This plexus partly joins with the lymphatics in the interlobular septa and partly goes direct to the bronchial glands. The lymphatics of the pulmonary pleura open by stomata into the pleural cavity. In the parietal pleura there is a superficial and deep plexus of lymphatics which communicate freely and on which there are stomata. The mediastinal pleura has peculiar lymph nodules described by Knauff, with blood-vessels like glomeruli and with lymph cells only in part covered by the epithelium of the pleura. Similar nodules, but without the same arrangement of blood-vessels, are present in the diaphragmatic and costal pleura. The pleural cavity is in direct communication with these nodules by so-called "pseudo-stomata."

Differences between lung of child and adult.—The chief differences between the lung of the child and that of the adult are that in the child the lobulated structure of the lung is more distinct, the connective tissue being more pronounced, and the alveoli of the newly-born child are from 0·03 to 0·06 millimetre in diameter (Kölliker), while those of an adult are from 0·05 to 0·1 millimetre. As growth continues, fresh alveoli do not appear to be added, the increase proceeding from an expansion of all the parts.

DISTEMPER IN DOGS.—The master of the Flint and Denbigh Hounds has given instructions that the hounds and puppies are to be inoculated with vaccinia as a precaution against distemper.

ABERDEEN MEDICO-CHIRURGICAL SOCIETY.—The annual meeting of this society was held on Nov 20th, when the following office-bearers were elected for the year:—President: Dr. John Gordon. Vice-President: Dr. J. Theodore Cash. Secretary: Dr. John Marnoch. Recording secretary: Dr. Ashley W. Mackintosh. Treasurer: Dr. William Findlay. Librarian: Dr. Peter Howie. Members of council: Dr. D. W. Finlay, Dr. D. W. Geddie, Dr. Rose, Dr. George Watt, Dr. A. Westland, and Dr. George Williamson.

Four Lectures
ON
THE NATURE, CAUSES, AND TREATMENT OF CARDIAC PAIN.

Delivered at the Medical Graduates' College and Polyclinic in London on June 16th, 23rd, and 30th and July 7th, 1902.

BY ALEXANDER MORISON, M.D., F.R.C.P. EDIN.,

PHYSICIAN TO OUT-PATIENTS AT THE GREAT NORTHERN CENTRAL HOSPITAL AND PHYSICIAN TO THE CHILDREN'S HOSPITAL, PADDINGTON GREEN.

LECTURE IV.[1]

Delivered on July 7th.

ON THE DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CARDIAC PAIN.

GENTLEMEN,—The diagnosis, in its widest sense, of angina pectoris includes not only the discrimination of varieties of that malady from one another but that of the syndrome of angina from other conditions which in some measure resemble it yet affect quite other structures than those involved in Heberden's disease. For this general diagnosis three chief points have to be borne in mind and usually serve to exclude ailments which can only be mistaken for angina when their indications are imperfectly regarded. These guiding points are: (1) the situation of the pain; (2) the character of the consequences of the pain; and (3) the fact as to whether or not the act of respiration, especially in its voluntarily exaggerated execution, is in any way impeded or tends to influence the degree of pain. The site of pain in all varieties of angina pectoris is, in almost all instances, chiefly in the anatomical situation with reference to the surface of those structures, lesions of which we have found to be associated with Heberden's disease. That site is essentially sternal or retro sternal, and most commonly some point between the level of the third rib cartilages and the ensiform process. Cardiac pain is practically central and in the situation indicated. It is rarely epigastric or over the manubrium sterni. The patient in an attack when asked to place his hand over the seat of pain lays it over the centre of the sternum, and only in the second place describes the brachio-thoracic or other direction of radiation, if such be present. This fact serves to distinguish angina almost always from sub-diaphragmatic pain, whether of a functional or organic character, in the higher abdominal viscera. With the character of the consequences of cardiac pain we have already dealt at length in discussing the clinical history of the disease. While the general collapse which is observed as a consequence of hepatic, renal, or other severe pain may bear some resemblance to that of the later stages of an anginal attack, the characteristic radiation of the pain in cardiac cases usually serves to indicate on well-known anatomical lines the source or seat of the original stimulation. Collapse due to non-cardiac pain, moreover, is usually of later supervention than when due to angina pectoris. The last point—namely, the freedom of respiration and especially the unimpeded character of forced or voluntary respiration—serves to distinguish between cardiac and other thoracic pains whether direct or reflex, which, by affecting the intercostal, diaphragmatic, or other muscles exercised in forced respiration, indicate the structures involved. "From the disease itself," as Heberden remarks, "those who are affected experience no difficulty in breathing, by which circumstance this breast-pang is chiefly distinguished."[2] The patient's respiration, like his pulse, may, indeed, be temporarily arrested while in the agony of angina, but if he be asked to take a deep breath he can do so without impediment to respiratory movement or increase of pain. These considerations, borne well in mind, will usually serve to guide us aright in the diagnosis of angina pectoris

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[1] Lectures I., II., and III. were published in THE LANCET of Nov. 1st (p. 1173), 8th (p. 1246), and 15th (p. 1306), 1902, respectively.

[2] Commentarii, p. 309.

from pain affecting structures other than the heart. Mistakes will doubtless occur and exceptional and perplexing cases be met with even when these points are remembered, but such exceptions only serve to prove the value of what is a practical and safe clinical guide in this important matter.

By the more particular diagnosis of angina pectoris I mean, not only the discrimination of so-called true from false angina, but the distinguishing of varieties of true angina from one another. If the first task be at times difficult, and even when every precaution to come to a right decision has been taken results occasionally in error, how much more frequently must this be the case when we essay the second. There are cases of "true" angina to witness the phenomena of which is to have them indelibly fixed in memory and on the same conditions presenting themselves again they lead to the recognition of that state with promptitude. The clinical histories of the varieties of angina which I have already related contain examples of such. But there are other cases which so closely, in some particulars, resemble those I have mentioned, that it is only by a close consideration of details that we can detect where the resemblance ceases. Even after every care has been taken, however, and we have formed, it may be, a comparatively favourable prognosis, error may occur. As I have already contended, true and false are unfortunate terms to apply to angina unless we have a clear notion of what we imply by their use. If we mean by the former to designate cases which always threaten, and, in the vast majority of cases, sooner or later destroy, life, while by the latter we denote cases which, whatever the degree of distress they exhibit, very rarely indeed or never kill, we may be justified for convenience sake in making this distinction. As, however, the pain of a fatal angina may not to all appearance be so excruciating as that in cases which recur frequently and disappear apparently for lengthened periods or altogether—although the fact of suffering, of angina, is indisputable and real in both cases—its mere degree cannot be taken as a measure of its seriousness. Dr. G. A. Gibson, in his recent lectures on the Nervous Affections of the Heart,[3] also expresses his objection to a distinction being drawn between varieties of cardiac pain as true and false.

The criteria which distinguish cases which are dangerous from those which are not are only to be gathered from a comprehensive view of the circumstances of each case—a view which comprises the whole of the clinical picture and properly appraises the diagnostic value of special points or details. A margin for error must be left even then, for there are probably cases of which we may say with the poet, "There is no name for that of which she died." It is, however, probable that the sex of the deceased to whom the Poet Laureate applied these words in his Human Tragedy would exclude angina pectoris. I say this is probable, for a comparatively small number of women undoubtedly die from an angina which may long have been regarded as a mere manifestation of a troublesome dyspepsia. This was the fate of an elderly woman whom I saw as an out-patient at the Great Northern Central Hospital. Sex, however, is an important factor in this relation and of two patients, one a man and the other a woman, who manifest symptoms of angina the former will with incalculably greater frequency be found to harbour the fatal variety of the affection. Although cases of fatal angina in early life have been recorded and even atheroma has been demonstrated on rare occasions in the "teens" or earlier, as in a specimen which I show you, if we exclude the consequences of syphilis and the general debauchery to which man is at times addicted in earlier life, Heberden's indication of the fiftieth year, the average period of the commencement of atheromatous wear and tear of the vessels, may be regarded as a valuable element in the diagnosis of cases which manifest cardiac pain. At a still later period, say between 60 and 70 years of age, to pronounce a man sound at heart and to prescribe gymnastics, wood-cutting, and so forth because of a well-preserved softness of radial pulse and the absence of detectable cardiac lesion is, in many cases, to court the disaster which overtook one of the cases the history of which I have related. To be over 50 years of age, then, and to evince angina is to suggest, though not to prove, the presence of the dangerous kind.

Heredity, again, has a certain influence and has to be reckoned as a factor in diagnosis. The father of my patient who died from musculo-spasmodic angina died similarly suddenly when about to leave his house to conduct a religious service, for he was a clergyman. The anginous family history of the Arnolds is common knowledge. When the neurotic disposition is unassociated with a proneness to vascular degeneration, in the production of which gout is a frequent factor, angina is more likely to be less dangerous and more transient than when these dispositions are combined, as they frequently are, in cultured families and in those who exercise intellectual professions. Woman, whose frequently much-enduring but more refined constitution than that of man makes her more often than the latter the subject of the less dangerous variety, is also an illustration of this fact. In the death-grip of true angina, moreover, the patient is usually stilled into quietude by his agony and feels that his summons to pass into the unknown has come. In the agony of so-called false angina he frequently, indeed, fears that death is imminent, but he manifests more excitement and energetically endeavours to break loose from the grasp that threatens to detain him. Yet it must be confessed that these general impressions, although they have a certain value, are not altogether trustworthy, and the man who off-hand, from such considerations, pronounces definitely for or against the presence of the dangerous or innocuous variety, as the case may be, may find himself sooner or later admonished by the event that wisdom lies in exercising the greatest caution in expressing too positive an opinion. Given a comparatively young subject with normal radial arteries, be they tense or otherwise, free from local heart affection or other organic disease, and without the history of that ubiquitous agent for vascular evil—syphilis—we shall in most cases be justified in looking for a transient cause of angina and the disappearance of the effects of such. On the other hand, given a man (or it may be a woman) past the meridian of life, or with the evidence of local disease, or having an indubitable specific history, we may be fairly certain that Death has not only beckoned the patient, but will in many cases return at no distant date and insist upon his following him.

Admitting, however, that we are in certain cases concerned with angina vera or the lethal type, is it possible to determine in any way the particular variety of the fatal malady with which we have to deal? For example, is the case before us one of coronary calcification with neuritis, or it may be aneurysm, or is it one of musculo-spasmodic angina? In considering the point I am aware that I am treading upon altogether disputable ground and desire to avoid any appearance of a presumptuous dogmatism, but, nevertheless, I wish to point out some considerations which appear to have weight. The age of two such patients may not help us very much, but it is a point not to be dismissed hastily. Taking 50 years as the average age for commencing atheroma in normal circumstances—the period at which the normal wear and tear of life begin to tell on the blood-vessels—it is unquestionable that those cases which ultimately reveal calcification of the coronaries may exhibit the first evidence of angina considerably earlier. It is unnecessary to refer at length to the classical case of John Hunter, the clinical details of which have come down to us from Sir Everard Home and have been studied with care and rendered still more current knowledge by Sir William Gairdner. Born in 1729, he had the first attack of gout in 1769, his first angina in 1773, his second attack in 1776, and a more continuous status anginosus was established in 1785 which ended in death in an attack in 1793 when he was 64 years of age. My own patient, whose case I have related, died at the age of 54 years after having suffered from angina for seven years but with progressive frequency in the latter portion of that period, and also died during a paroxysm. During the 10 years prior to his death he had four times suffered from gout in the great toe. Both these cases, therefore, must early have manifested coronary atheroma and probably, likewise early, the calcareous change in the vessels. In the case of my own patient I explain the earlier symptoms by neuritis and the later and more continuous by neuritis and aneurysm. Time alone can show whether this conclusion, which is probably correct for the case in question, is of more general application.

The sufferer from musculo-spasmodic angina may quite possibly be affected as early as the patient from coronary neuritic angina, but in all probability, in such a case, some sufficient cause of muscular exhaustion will be found in the events immediately antecedent to the attack which may point to the muscular factor playing the major rôle in the tragedy. The arthritic history may likewise be absent, although not necessarily. But, questionable as these criteria

[3] Edinburgh Medical Journal, 1902.

may be, I think there is a less doubtful value to be attached to the general type of the sufferer. The canvas of Sir Joshua Reynolds, painted in 1787, and Sharp's fine engraving made at the same date after repeated attacks of the malady had probably borne in upon the mind of the great student the true significance of his pain, have brought down to us the keen, alert, and cogitative face of Hunter, associated with a body naturally powerful and, although short, certainly not stout. Those, on the other hand, who possess the heart most prone to musculo-spasmodic angina—the fatty heart—usually exhibit a general *embonpoint*. They are usually plump of face and stout in body, a stoutness in which the internal organs participate. Till now we have had to be content with such general impressions as these for the purposes of diagnosis. But are we to-day at the end of our resources when we have considered these? The fortunate accident which threw the shadow of the bones of Roentgen's hand on to the photographic plate—and how frequently the enlightening incident is an accident—seems to point to a possible solution of the difficulty on more reliable lines.

I shall presently show you on the screen a skiagram of the heart of my own patient made after death (Fig. 10). It

FIG. 10.

Skiagram of calcareous coronary arteries. The vessel with descending branch to the left is the left coronary artery.

was placed upon a celluloid plate, the light was centred over the coronaries, and it was given a short exposure of about one minute and another of four minutes. The result was equally, or nearly equally, demonstrative in both instances of the ease with which the x ray differentiates between the calcareous arteries and the surrounding textures. Such arteries in the leg and arm have been demonstrated during life and it does not seem improbable that sufficient, if not equal, success (for the organ containing them is mobile) may one day attend its use when examining the heart laterally at the level of the coronary circle. This lateral method of examining the heart naturally suggests itself, and I find that Dr. F. H. Williams in his recent work[4] draws attention to it in some detail. The limited trial I have myself made of this method in connexion with an attempt to see clearly the base of the heart inclines me to think that until some further development in the re-focussing of the rays after they issue from the Crookes's tube is made, more success is likely to attend radioscopy with the fluorescent screen than radiography. But even if by this means we cannot at present determine the existence of calcareous change in the coronary arteries themselves there is no difficulty in detecting such a condition if present in the more stationary vessels of the limbs. Just as we examine the tension and character of the pulse by palpation for diagnostic purposes, so also by skiagraphy we may determine whether or not the general arterial change in a patient is associated with calcareous deposition. Thus, by an argument from the general to the particular, we may attain to a reasonable estimate of the condition of the coronary arteries of the heart, even though our conclusions may not rise to absolute certainty. Dr. Williams[5] also states that both by radioscopy and radiography calcification of portions of the ascending aorta may be detected. Fig. 11,

FIG. 11.

Skiagram of the left leg of an old man, showing osseous tumour of the tibia and calcareous arteries. A portion of the anterior tibial only is seen in this print, but the photograph showed the vessels well.

which demonstrates calcification of the anterior and posterior tibial arteries in an old man, I found in Messrs. Coxeter's collection of prints. The case appears to have been skiagraphed on account of the osseous tumour of the tibia, the arteries revealing themselves accidentally.

That the detection of calcareous arteries in the heart or elsewhere would be of importance is unquestionable, for dealing as we probably should be, in a case with such vessels, with a fairly sound muscle, and in cases with non-calcareous arteries with a heart invaded by fat or degenerated in texture, our prognosis would be influenced by these considerations. After all the essential organ of circulation is the cardiac muscle, not the cardiac blood-vessels, and so long as the former is capable of exercising its function it is only the accident of pain arising in the latter which can bring the heart to a standstill. The case may last for years, even if in its later phase the patient should suffer much. But he who carries a degenerated heart, an enfeebled muscle, and manifests true angina is not merely at the mercy of an accident. He need not suffer pain, but he may succumb to a syncope when least expected. Nay, the very absence and comparative rarity of pain may lull him into a false security, in which he may place more weight upon the degenerated fibre than it can bear. The history of many a musculo-spasmodic angina from fatty heart is that of an organ assumed to be healthy and of its owner using it accordingly. It is, of course, possible that calcareous degeneration of the arteries may co-exist with fatty degeneration of the heart, but there is at present a lack of precise information on this point; for that minuteness of detail in examination necessary to establish the fact is wanting in the record of most cases. Even John Hunter, according to Edward Jenner,[6] overlooked the condition of the coronary arteries in a case of angina pectoris which was under the care of Heberden and I am not aware of the account of any case of angina in which the condition of the general arterial system has been examined, even when the state of the coronaries is fully dealt with. That it

[4] The Roentgen Rays in Medicine and Surgery, New York, 1901.

[5] Op. cit., p. 385.

[6] Parry's Syncope Anginosa, p. 3.

would be well that a general inspection of the arterial system should be undertaken in all such cases, clinically and pathologically, can scarcely be gainsaid. For a close scrutiny of the particular and a wide view of the general is admittedly the most fruitful habit of mind for the scientist. That way lies the perception of law.

The symptoms of acute aortitis, as we have learned from the clinical history of the condition, are sufficiently diagnostic and taken in connexion with the comparative youth of the sufferers and the frequent history of specific infection enable us to distinguish with some confidence this form of angina from others. Even in its more chronic phase, youth, syphilis, and the locality of pain serve to guide us in coming to a conclusion. In those cases of angina associated with aneurysm of the aorta, when other well-known criteria of this condition fail us, Roentgen radiography is invaluable. Indeed, in no sphere in which it has been employed has its value been more generally acknowledged than in this. Neuritis being a concomitant of other conditions its recognition will be regulated by the diagnosis of those states with which it is associated, but the determination of its intra- or extra-vascular seat must at present be left to the pathologist.

The guide to the discovery of endocardial angina is the presence of valvular lesions and it is questionable whether intra-cardiac pressure plays more than a very subordinate part in these cases. That it plays some part is probable, for the reasons already given, but I feel disposed to think that its influence has been over-estimated. Even in the dextral valvular case I have mentioned there was an endarteritis at the commencement of the pulmonary artery which cannot be ignored as a factor in the production of pain, under the flapping stroke of the large vegetations which were attached to the valves. That the pulmonary artery, like every other artery in the body, is innervated, I satisfied myself when preparing a course of lectures on the Relation of the Nervous System to Disease and Disorder in the Viscera.[7] The syndrome of angina, coupled with various forms of peripheral dysæsthesia such as coldness of the hands and feet, disturbances of sensibility generally, and at times of motion, unassociated with valvular disease and without evidence in cardiac muscle or in blood-vessel of any of those persistent changes which we have seen to be associated with the graver varieties of the complaint, is in all probability purely vaso-motor and of favourable augury.

That most cases of angina pectoris are of compound not simple origin has been already argued, and the discrimination of one variety from another is largely merely a matter of emphasising what may appear to us to be the "predominant partner" among several factors. The absence of pain and the syncopal signs in a large number of cases of angina sine dolore will serve to distinguish this state from Heberden's disease.

THE PROGNOSIS OF ANGINA PECTORIS.

If we regard angina pectoris as one and indivisible, our prognostication of the probable issue of any particular case will be based on such a mass of dissimilar data that our prophecy, always uncertain in angina vera, to use the old collective term, must of necessity lack even a rational degree of precision. As I have endeavoured in the preceding lectures to classify provisionally the varieties of angina, both organic and functional—to employ terms having a conventional meaning—I shall consider, in the fewest possible words, the prognosis of angina on those lines, even although the future to which we must all bow should pass a sponge over the whole slate which I have had the temerity thus to score. If we have reason to suppose that a given case is the subject of coronary vascular angina, neuritic or aneurysmal or both, how can we hold out hope of recovery from it or an amelioration of its symptoms? The process we are dealing with we know to be chronic and progressive. There is a curious rapidity in malign progress in the later phases of the disorder, as though a fresh aggravating element had been introduced. This may or may not be aneurysmal in character. That it probably sometimes is will not, I suppose, be questioned in view of the facts I have related. In the earlier and less urgent phase of this variety, then, we may guardedly and, viewing the patient as a whole and not as a coronary artery, hold out the hope, under judicious management, of a considerable length of life even though we cannot remove the sword of Damocles to a point where it may harmlessly snap the thread which suspends it. But when the progressive degeneration of the vessel has called into painful activity the slumbering sensibility of the coronary nervous system it will require little foresight to foretell a fatal issue in the near future whether the patient himself be made the participant of our belief or no. It is rarely that the physician is so fortunate as Dr. Bucknill was who had as a patient Thomas Arnold of Rugby. "He next asked," writes Dr. Bucknill, "if the disease was generally fatal. I said 'generally' (for those who knew him were aware that it was impossible not to tell him the exact truth)."[8] Thomas Arnold, however, had not, as we know, calcification of the coronaries, but probably fatty degeneration of the heart, for its external surface is reported to have been healthy, its walls thin, and the colour of the muscle pale and brown. He died in his first attack of what, according to the classification I have attempted, would be called "musculo-spasmodic angina."

What is the prognosis in this variety? A man, unconscious of any failure of power, strenuously occupied bodily and mentally, is suddenly attacked by angina and the heart, overwhelmed by pain, ceasing to beat life is extinct. Is there any reason why, if a patient recover from the actual paroxysm of such an attack, the heart muscle, which did its work so well the day prior to his death, should not continue to do it equally well if he survive the attack, provided the danger-signal raised by such an event be borne in mind? We know that death in a first attack of angina is not common whatever the underlying organic cause of the affection, but fulminant cases are usually associated with a state of cardiac muscle which precludes what Latham describes as the "strenuous exercise" to which "T. A." was addicted, and the continued life of such a patient must be contingent, if he survive the paroxysm, upon the abandonment of such. Any prophecy as to its duration must depend upon the impression made upon the physician of the degree of underlying cardiac degeneration which the attack has revealed. There is more hope for the degenerated heart of a fat man, speaking generally, than for the degenerated heart of a thin man. In the case of the former we shall with greater probability have to deal with fatty infiltration and a possibly healthier state of the muscular fibres of the heart than in the latter, in whom the muscle itself is more probably affected with fatty degeneration.

The prognosis of acute syphilitic aortitis must be dependent upon the extent of the affection. An extensive inflammation of the aorta associated with angina may kill, whatever its cause. The acute phase of this condition may be regarded in some sense as we do the paroxysm of angina in the cases we have already considered. Once safely over the acute stage of such an aortitis the patient, especially if his condition be rationally referable to syphilitic infection, frequently responds well to specific treatment. The angina which has been the plague of the patient and the despair of the physician often ceases to trouble, to the gratification of both, when anti-syphilitic treatment is prescribed and a favourable prognosis is accordingly justified in such a case. The prognosis of aortic aneurysmal angina is the prognosis of aneurysm, but as regards the anginous element in acute aneurysmal cases I have already given reasons for believing that pain is more characteristic of the intra-arterial than of the extra-arterial stage of its growth. The prognosis of neuritic angina has also already been considered. It is the prognosis of the probable effects of the neuritis in the situation in which it occurs, be it coronary, aortic, or extra-vascular. Inasmuch as the diagnosis of neuralgic as distinguished from neuritic angina is frequently established by the recovery of the patient it is scarcely necessary to add that its prognosis is favourable. This is also true of so-called vaso-motor angina and of anginæ reasonably referable to other than cardiac reflexes, such as poisons, tobacco, or other. "*Cessante causâ cessat et effectus.*"

The severe cardiac pain associated with valvular lesions, and particularly as has been stated with aortic lesions, being probably due to the disordered action of the organ in a condition of non-compensation or of lost compensation, and more precisely to a sudden systolic distension of the base of the aorta and of the coronary vessels, it is to be expected that on the attainment or restoration of compensation the anginal symptoms will diminish or pass off. This, the history of many of these cases, justifies us in predicting. In using the term "distension" in this relation I do not mean

[7] Edinburgh: Pentland, 1899.

[8] Latham's Works, vol. i., p. 455.

spasm, like Heberden, if he be correctly believed to have used "*distentio*" in this sense, but pain from stretching. In this connexion it is of interest to note that while valvular heart disease is, as we know, very common in children, the vaso-motor phenomena associated with these lesions which we so frequently see in older subjects are among children very rare. That they occasionally occur is probable, but I confess I have never witnessed a case, say, in children under 12 or 14 years of age. Heberden states that he met with angina in a child, aged 12 years. This was probably an endocarditic case. The comparative immunity of children from this symptom seems to argue their escape as being due to the greater elasticity of young tissues, and is thus an argument in favour of the view I have expressed as to its *rationale* in older subjects. The prognosis as to pain, then, in most of these cases is probably to be guided by the prospect of the establishment or restoration of a due balance in the circulation by improved compensatory growth of the organ. The prognosis in compound cases, a class which probably includes the majority, must be governed by the prospects of that factor which is assumed or believed to play the major rôle. Angina sine dolore being essentially due to cardiac muscular failure the prognosis in this type of the affection is of the gravest. It is a musculo-paretic, not a musculo-spasmodic, condition, hence, possibly, the absence of pain. The patient is less at the mercy of an accident such as pain, but the cardiac muscle itself is near the termination of its vital possibilities.

We have now discussed the nature and causes of cardiac pain with the imperfection which is, in the present state of our knowledge, unavoidable. I fear, indeed, that I may have appeared to speak on some points with an emphasis unwarranted by the facts before us, but my action has been dictated by the belief that it is by seeking an organic basis, not of one but of several kinds, that we are likely to obtain precise knowledge of a subject which has been largely dealt with as an appalling symptom, and for the explanation of which some authors like M. Henri Huchard offer us only one key. We shall find in considering the treatment of angina pectoris that we possess no panacea. It is, I believe, equally true that for the explanation of the malady we possess no single key; but that there are groups of cases the nature of which, however imperfectly explicable at present, will in time by patient, systematic, histological investigation be explained on more than one hypothesis; for pathological histology is the anatomy of structural disease.

THE TREATMENT OF ANGINA PECTORIS.

If the life be more than meat and the body than raiment, it is likewise true that the possession of life is more than a knowledge of its mechanism. The wisdom of the physician is based upon impediments to the manifestation of life and disaster attending its exercise. The physician is in a minority among men, whether as belonging to a recognised calling or profession, or whether the term be used metaphorically, as applicable to one who has learned from his own limitations to live within the compass of his powers. Notwithstanding instances of disaster to individuals it is on the whole perhaps well that caution and prudence, as regards the preservation and management of life, have not characterised mankind as a race. Many a heroic deed, many a strenuous life lost in the endeavour to reach a high ideal, would have been left unrecorded if the hero, possibly an invalid, had known and endeavoured to obey the dictates of physical prudence which such wisdom would have inculcated and had not with both hands thrown away his life for something better than life. John Hunter, who knew the serious nature of his malady, did much of his best work after angina pectoris had marked him for its own. But with such general considerations, which may be safely left to take care of themselves like the rising and the setting of the sun, the physician who has before him a patient who has, or may have, angina pectoris is in no way concerned. The bond between him and his patient is not the potential heroism of the latter but the possibility of his demise if the dictates of physical wisdom be not obeyed, and in many cases of angina, unfortunately, whether they be obeyed or no.

Let us consider first the case of the patient who *may* develop angina pectoris. He is probably a man past the meridian of life, either spare, wiry, and it may be with a history of gout, or stout, aging, and perhaps with some history of occasional circulatory disturbance such as giddiness or breathlessness on exertion. They have both probably consulted us about some other matter altogether—dyspepsia or what not—and they have both, also probably, a degree of arterial atheroma even though the radials at the wrists or the more accessible arteries should not afford an indication to this effect on palpation. To auscultation their hearts may be perfectly sound. Let us beware in such circumstances when prescribing rules for the general conduct of their lives, dietetically or otherwise, not to allow them to go away with the impression that there is no particular need for caution in the general expenditure of their energy. The man, 50 years of age, who wishes to reach 70 years without the possible introduction of an angina into his life has to adopt an old Scottish motto, "Gang warily." The failure to inculcate this advice as regards physical exercise, especially if unwonted restrictions be placed upon the patient's diet at the same time, is, I believe, a fruitful cause of some of the fatal attacks of angina we meet with. As a corollary to this conclusion it may be added that caution in the resumption of activity, especially in those of atheromatous age, during convalescence from debilitating ailments, influenza and other, is of the first importance in this connexion. This is especially necessary if such patients should ever have manifested any indication either of angina cum dolore or of angina sine dolore. A little more patience and caution exercised in this respect and many a valuable life might have been prolonged for a shorter or longer period, to the advantage alike of individuals and of those dependent upon them. If we inculcate such precautions in the case of one who appears to us to be a candidate for angina, how much more will it be our duty to do so in the case of those who have actually experienced an attack? We must in such cases not only indicate a suitable dietary and inexorably limit the exertion of the patient to such an amount as his own experience teaches him is safe, but also point out to him the danger of emotional excitement and the comparative safety of a calm and equable state of mind. The desirability of the attainment of this calm may be indicated by the physician; the world is too old for it to be necessary for me to add that every man must build that inexpugnable citadel of his existence for himself. Into the details of the dietary of these cases I need not enter fully because they must be regulated according to the type of angina we have to deal with. We may have to advise one man to become a vegetarian and another to eat meat, but a strict moderation as regards the quantity both of solids and fluids must be prescribed, as we are dealing with a condition very sensitive to fluctuation in blood pressure. For this reason, also, the clothing and climatic surroundings of the patient have to be regulated. Those who in their physical misfortune have the consolations of wealth should avoid the rigours and charges of an unsettled climate by living during the more severe weather in more favoured portions of the globe. Those who cannot do so must, so far as possible, by loose warm clothing, and especially sufficiently warm clothing in bed, endeavour to create that summer in their own surroundings the attainment of which elsewhere their circumstances do not permit them to seek.

As a transitional stage to the consideration of medicinal agents, invaluable in some phases of the disease and useful in others, let us shortly deal with the question of baths and exercises. In connexion with angina pectoris the differences in the operation of these two processes must be borne clearly in mind. Long known and universally employed as has been medical bathing it is to Nauheim that attention has been chiefly directed since Beneke wrote on the effects of hot salt-water bathing on chronic heart disease. His writings on the subject appeared at intervals between 1859 and 1875 and they are those of a cautious and candid observer. He states in a pamphlet published in 1872[*] that while many of his cases improved after a course of treatment there was little immediate effect observed upon the action of the heart and on the character of the pulse. This observation by Beneke agrees with my own experience as regards cautiously employed warm baths. It is reported of Lord Beaconsfield that, on being asked by an ambassador of his party who had received an invitation to dine with a political club of the Opposition whether he might accept the invitation, the Premier answered, "Why not? a man must dine somewhere." To us sons of the Norse water-dogs, who still smell the brine dashed from the prows of their

[*] Zur Therapie des Gelenk-Rheumatismus und der mit ihm verbundenen Herzkrankheiten.

war ships in the faces of our Viking ancestors, it is at least as desirable that a man should bathe somewhere. And whether he dine or bathe, so long as he do so prudently, the effect is calculated to be beneficial, provided he be not too much exhausted either to eat or to wash. If, when sufficiently able to do either, he do both regularly and systematically for a month or so, and thereafter rusticate in a pleasant atmosphere and take gentle exercise for an additional period far from the madding crowd and the harass of his calling, it would require a very pessimistic person to prognosticate that he would not return refreshed from his holiday to face the duties of life. Such a course of treatment is not calculated to be detrimental to any circulatory disturbance whatever or to any other form of physical debility. In a case of the type in which sclerotic angina frequently occurs and which I examined at Nauheim in 1896, as may be read in my book on "Cardiac Failure and its Treatment,"[10] I found that the vascular changes induced by recumbency in a Nauheim bath differed little from those induced by recumbency on a Nauheim sofa, for the patient visited me at my hotel on the same day upon which I examined him in his bath. I do not wish to imply by this statement that we cannot by manipulating the temperature and contents of a medicinal bath powerfully influence the circulation, but merely that a bath of moderate temperature and of not too irritating constitution, such as is usually employed, is rarely detrimental to any kind of circulatory disorder. My judgment on this point is upheld by the practice at Nauheim to-day, for whereas at one time practitioners there considered that the baths were not suited to arterio-sclerotic conditions they now believe that with circumspection they may in such cases be used without injury to patients. In short, given the presence of organic angina pectoris, the bath which will not injure the patient may be harmless but cannot be rationally expected to be beneficial.

I have had occasion in the course of these lectures to dissent from some of the conclusions at which that distinguished French physician, M. Huchard, has arrived as to the nature of angina pectoris. I am glad to find myself at one with him as regards the balneological treatment of this class of case. "It is not necessary," writes M. Huchard,[11] "to share the enthusiasm without doubt disinterested, of the German author who claims to cure true and organic angina pectoris by the help of these waters. I have seen, indeed, some of his patients really cured, but they were cases of false angina of which the precise diagnosis had not been established and which ought to have been cured by any other means. I also saw a case of true angina who, curiously, resisted a species of therapeutic suggestion exercised upon him and who returned to France in considerably worse plight. This pseudo-success is not to the credit of the waters of Nauheim." But the case is far otherwise with the exercises whether mechanically or manually administered and which are at times of much service in appropriate cases of cardiac debility or disordered compensation in valvular diseases of the heart. Of these methods there can be but little doubt that the Swedish resisted movements systematised and regulated in their application to cardiac cases by the late Dr. August Schott are the safest and best. But be they never so safe and never so good in suitable cases, the increase in the force of cardiac systole which results from their employment places them entirely out of court in the treatment of any but the most innocuous forms of angina pectoris. Remembering the possibility of the occurrence in coronary angina of such an aneurysm as I have demonstrated to you, I ask whether any rational being would advise any resistance movements in such cases with the knowledge that such a lesion may underlie the manifestation of pain. I place no mark of interrogation after this sentence for it requires no answer. An occasional anginous attack in one probably the subject of fatty heart may not, perhaps, be capable of being placed in the same category as those I have referred to, but the advisability of either baths or exercises even in such a case, provided there be evidence of the attacks having been indicative of an organic basis to the symptom, is highly questionable.

If on account of a possible aneurysm in connexion with angina exercises of the kind mentioned be contra-indicated, what is to be said of complete rest? We know that angina may occasionally be a troublesome symptom in aneurysm of the aorta; we know also that absolute rest, especially if associated with a reduction of the quantity of the food taken, on the lines practised by Tufnell of Dublin, has resulted in amelioration of the symptoms due to aortic aneurysm. Would these results indicate the employment of a similar line of treatment in cases of coronary angina even if we had the means of determining positively that the physical basis of the disease was aneurysmal? There is no doubt that complete rest is less frequently associated with attacks of the pain than activity. The conditions of the blood pressure, therefore, in this state are more equable than during movement. On the other hand, the patient, possibly a gouty man and but for his attack a healthy man, condemned to his couch for an indefinite period, would be quite likely in his enforced inactivity to manifest signs of his constitutional taint. If we grant that with careful supervision and a restriction of food to the necessities of his body such a disturbance as an attack of gout be avoidable, could an attempt to induce coagulation in the very small sac of an internal coronary aneurysm be brought about without the greater evil of a coronary thrombosis? In discussing the prognosis of angina pectoris Dr. Osler[12] states that "recovery is quite possible, and there are instances in which the attacks disappear entirely," but, unfortunately, the instance he mentions was but an angina symptomatic of lost compensation in aortic valvular disease. That such recover with the restoration of compensation in many cases we know. I am not aware, however, that there is any record of the recovery of a case of coronary angina, even though it may have been slow in development and compatible with life for a lengthened period. It is theoretically possible that a small and even dissecting internal coronary aneurysm might so thicken and shrink as to become practically obliterated with or without blood coagulation in its interior, but such a case has to be found before this supposition can be substantiated. That it has not been found may, perhaps, be due to its not having hitherto been sought for or suspected to exist. I have urged these theoretical objections against complete rest in coronary angina that I might not appear to lose sight of the collateral evils which may attend any special course of treatment. But were the dangers of coronary thrombosis in the circumstances very great, they might be equally urged against the treatment of aortic or of any other aneurysm by this method, for coronary disease might be coincident with these, and the occurrence of coronary thrombosis would probably bring about the death of the patient. If, therefore, much judgment would be required in instituting treatment by complete rest in the early stages of a coronary angina there need be no hesitation in asserting that in its later phases as indicated by an increase in the frequency of the paroxysms, rest is the only rational treatment of the disease. It is calculated to postpone as long as may be the final scene, and it is not impossible that by some fortunate accident it might induce greater defensive powers in the artery which might cure the patient. The possibility of such a cure we may admit; the prospect of it we are not at present justified in holding out to our patient. The fear of inducing thrombosis by recumbency in the coronary arteries need not, however, deter us from instituting such a treatment, for, as Dr. G. W. Balfour has pointed out, the coagulation of blood, even in aneurysmal sacs of large size, is not easily brought about during life, and improvement when it occurs under rest and the use of iodide of potassium appears to be induced rather by a thickening and shrinking of the walls of the aneurysm than from coagulation within it.[13] In the internal coronary aneurysm I have shown you the blood coagulation in the lumen of the artery and that in the sac were evidently both of about the same age and probably both occurred either in the act of death or very shortly before it.

The drug treatment of angina pectoris is divisible into (1) the management of the paroxysm of cardiac pain and (2) the medicinal treatment of the patient in the intervals between attacks.

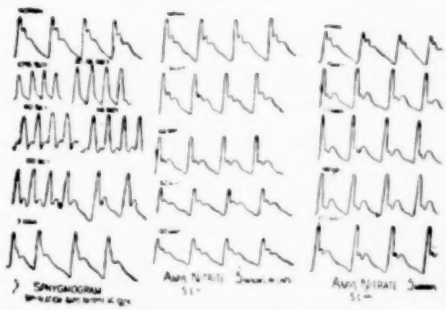
1. *The treatment of the paroxysm*.—The mine once laid, be it in coronary, cardiac, or aortic lesion, the spark necessary to its explosion is, in a large number of cases, exaggerated blood pressure, whether direct and propulsive from the heart or indirect and obstructive from the peripheral arterial system. This is probably the case in all save the musculo-spasmodic and musculo-paretic or syncopal cases. It follows that in most cases the regulation and

[10] Rebman, London, 1897, p. 129.
[11] Maladies du Cœur et des Vaisseaux, op. cit., p. 420.
[12] Op. cit., p. 139.
[13] Lectures on Diseases of the Heart, p. 467.

moderation of blood pressure are the primary objects which the physician has in view when he prescribes a medicinal agent to remove the injurious effects of excessive vascular pressure. Concurrently with these it may be also necessary on occasion that he should employ more direct analgesics, and in the final stage, if need be, one form or another of artificial respiration or cardiac stimulation, to which reference may now be made. Cardiac failure in angina pectoris being primary or due to vagal inhibition, as has been maintained, the employment of Laborde's method of rhythmical tongue traction[14] in combination with Silvester's method of artificial respiration is indicated. In the profound and prolonged syncope observed in the status epilepticus I have repeatedly found Laborde's method apparently very efficacious. Laborde's own explanation of the *rationale* of this procedure seems sufficient. He attributes its efficacy in great measure to the stimulation by tongue traction of the lingual fibres of the superior laryngeal branch of the vagus and to the cardiac and respiratory reflexes thus provoked. The combination of artificial respiration with tongue traction, which might be termed the Silvester-Laborde method and for which the coöperation of two persons is necessary, would most completely fulfil the indications in anginal syncope. But the direct heart-stab of angina pectoris which has reached the final stage of cardiac inhibition will probably prove more hopeless of recovery than syncope from other causes. There is no valid reason, however, why perseverance should not occasionally be rewarded in these circumstances as well as in others in which such persistent efforts to restore animation have been successful.

The most powerful influence in counteracting the paroxysm of angina pectoris must be ascribed to the *nitrites*. Nitrite of amyl, the vaso-motor influence of which was originally determined by Guthrie in 1859,[15] was first used in the treatment of this disorder by Lauder Brunton in 1866. Whatever difference of opinion may be entertained regarding the physiological mechanism of angina pectoris there can be none concerning the frequent efficacy of the nitrites in relieving cardiac pain. In 1879 Murrell[16] enriched our therapeutic armoury by using nitro-glycerine in the treatment of angina pectoris and Matthew Hay in 1883 used nitrite of sodium for the same purpose. One of the best general accounts of "the pharmacological action and therapeutic uses of the nitrites and allied compounds" which has appeared in the English language is that given in his Croonian Lectures by the late Dr. D. J. Leech[17] of Manchester. The scope of my present lectures precludes a digression into the very interesting pharmacological and physiological phenomena revealed by a study of the effects upon the circulation of the nitrite group of medicinal agents and I cannot do better than refer those who wish to peruse a masterly treatment of the whole subject to the lectures of Dr. Leech, an observer whom medicine could ill spare. There are, however, some points in connexion with the comparative celerity with which these drugs act and the length of time for which their effects last which are of practical moment in the treatment of angina pectoris. These points have been well summarised by Dr. Leech[18] in the following words: "Changes in arterial tension are not often indicated by subjective phenomena and their extent and duration can only be measured by the sphygmograph. With this instrument I have tried by means of tracings taken frequently to estimate the time at which it reaches its lowest point and its duration. The influence of amyl nitrite on the pulse commences a few seconds after inhalation (Fig. 12). The tension is reduced to its lowest point in from 50 to 60 seconds and remains extremely low for 30 or 40 seconds, the pulse waves being reduced in size and sometimes irregular. Then it rises again, occasionally suddenly, and in from a minute and a half to two minutes the pulse is only a little lower in tension than it was before the inhalation. The slight lowering may continue for several minutes. Isobutyl and isopropyl nitrites when inhaled act almost like amyl nitrite. Sir B. W. Richardson, to whom we are so much indebted for knowledge concerning amyl nitrite, has drawn attention to the fact that when it is taken internally its effects are much slighter though more prolonged than when inhaled. Instead of the tension falling at once and recovering in two minutes it falls gradually for usually 20 to 25 minutes, remains low a short time, and then rises to its normal height an hour to an hour and a half after the dose has been taken. Because amyl nitrite is such a powerful agent when inhaled one drop is regarded as the proper dose but this is a mistake; from three to five minims may be required to produce about the same effect as that which follows two grains of sodium nitrite. A small dose of sodium nitrite (two grains) distinctly affects the pulse in two or three minutes; the point of lowest tension is usually reached in from eight to 40 minutes and distinct influence on tension ceases in one to three hours. Ethyl nitrite has much the same effect and on the whole lasts as short a time." The sphygmogram which I have copied from Dr. Leech's third lecture[19] illustrate some of these points (Fig. 12). The tracing which I also now show you from the pulse of the patient suffering from angina whose case I have related illustrates the effect on the pulse of the nitrites (1) in an interval between attacks and (2) during the attack (Fig. 8). They were published in *Treatment* of Oct. 14th, 1897. A shows the character of the pulse when the patient was free from pain and not under the influence of trinitrin. A dose of trinitrin was then administered to him with the results shown in B. The upper of the two tracings in this figure shows the acceleration of pulse and depression of pressure characteristic of the action of the nitrite on a pulse not under the irritative influence of pain and its gradual rise of pressure as the accelerant influence of the drug passes off. C shows the character of the pulse taken after an attack had just passed off and during which no trinitrin was taken. In D the lower of the two tracings exhibits the quick, small, hard pulse characteristic of some cases of angina which may be instructively compared with the unirritated yet accelerated pulse due to the action of trinitrin in the absence of pain, as shown by the upper tracing in B. The upper of the two tracings in D is characteristic of the pulse when an attack of angina, combated by the use of the nitrite, is passing off. With these tracings from a classical case of Heberden's disease we may also profitably compare those I have already shown you when discussing the *rationale* of endocardial angina and which are from a case of regurgitant disease of the aortic valves. They show the small, accelerated, low-tension pulse of some cases and the gradual rise of pressure in the pulse as pain passes off and the artery is filled under the influence of the nitrite (Fig. 9).

FIG. 12.



Tracings showing the relative effects of (1) the inhalation and (2) the internal administration of amyl nitrite; and (3) of the internal use of amyl nitrate. The upper tracings are the normal, the lowest after the lapse of a considerable period (one and half to three hours), and the intermediate of the effects of the drugs from 10 to 100 seconds after the exhibition (Leech).

As a rapid action of the drug is desirable during an attack of pain the inhalation of the nitrite or the internal administration of trinitrin in a fluid form is indicated. Of these the method by inhalation is more prompt in action and is to be preferred in the circumstances. Nor need we fear the

[14] Les Tractions Rhythmées de la Langue, &c., par J. V. Laborde, Paris, 1894.

[15] The Quarterly Journal of the Chemical Society, London, 1859.

[16] THE LANCET, Jan. 18th (p. 80) and 25th (p. 113) and Feb. 1st (p. 151) and 15th (p. 225), 1879; and Nitro-glycerine in the Treatment of Angina Pectoris, London, 1882.

[17] THE LANCET, June 24th (p. 1499) and July 1st (p. 3), 8th (p. 76), 15th (p. 135), and 22nd (p. 177), 1893.

[18] THE LANCET, July 1st, 1893, p. 3.

[19] THE LANCET, July 1st, 1893, p. 5.

use of more rather than less than the conventional dose of five minims of nitrite of amyl, for it has, accidentally and intentionally, been demonstrated over and over again that although a very small quantity of this drug has a physiological and therapeutic effect, very large quantities have been inhaled and used without persistently ill effect. Even the 1 per cent. solution of nitro-glycerine may be taken by some patients in considerable doses, though others can only tolerate small doses. Dr. Murrell mentions a case which took 100 minims of the solution as a dose, while another suffered severely from half a minim.[20] I generally use two to five minims and the only inconvenient consequence I have noted has been some degree of headache. The dioxidation of the blood under the influence of the nitrites may be disregarded for therapeutic purposes as the effect of these drugs is very evanescent. But gratifying as is the result of the use of the nitrites in many cases of angina they do not by any means always succeed in relieving the patient. The agony may continue notwithstanding their free use. In these circumstances, placed as we are between the possibility of witnessing a fatal inhibition or syncope and our own fears lest we should use too powerful an analgesic, we must run some risks if necessary to arrest an almost certain catastrophe if the pain should continue. Our sheet anchors then are chloroform, with or without the subsequent or combined use of ether and the hypodermic injection of morphia. On confronting such a situation Dr. G. W. Balfour writes as follows[21]: "In a few cases the relief obtained (by the use of nitrites) is sudden and complete; more often the relief that follows is gradual and doubtfully due to the treatment, while in a few cases no relief seems to follow even the most prodigal use of these drugs. Then we are forced to have recourse to the free administration of chloroform and this must be given so freely as to narcotise the patient rapidly and completely; given in this way I have not seen any case that has not been relieved, though I have seen several in which the relief has not been permanent enough to restore the patient to comfort. In these cases the chloroform has had to be supplemented by the hypodermic injection of morphine, and of this I have never hesitated to give a sufficient dose, generally from half a grain to a whole grain; such a dose as this has kept the patient asleep for some hours and he woke free from pain but exhausted." In view of the opinion which I strongly hold, that in the fatal issue of painful angina inhibition of the heart exercised by way of the pneumogastric nerve plays an important part, it is desirable that the use of atropine should be combined with the employment both of the nitrites and opium and other remedies—indeed, that it should always be used in these cases. For we know that, experimentally, the inhibitory function of the vagus is placed in abeyance by the use of atropine and its influence in small doses upon the heart is at once to accelerate its rate and augment its force. I should add that Dr. Leech disapproved of the use of morphia. Even in the later stages of angina he states that increased doses of liquor trinitrini up to 20 minims is a safer proceeding.[22] The method, degree, and rapidity of anæsthetisation must, however, be gauged in each case. It may safely be stated that the experience of no one man will include a very large number of cases of organic angina pectoris (other than those connected with valvular disease) requiring such extreme measures. Personally I have only met with three such. Stokes, one of the founders of cardiology, if we may use the term, confessed, as I have stated, that he had never seen even a mild case of angina other than that kind of cardiac pain associated with valvular lesion, and this will rarely require such vaporous anæsthetics as chloroform, although it may often require the hypodermic injection of morphia in addition to the use of the nitrites. This combination has also been found useful by Huchard. In short, cardiac failure, threatened by cardiac pain, is to be treated on the same lines as cardiac failure threatened by hepatic, renal, or any other form of physical suffering. It must be remembered, moreover, that the angina associated with valvular disease (usually aortic) is frequently in direct ratio to the degree of non-compensation in the ventricle and that production or restoration of compensation is the only road to a permanent removal of the angina. While, therefore, the important but engrafted symptom of cardiac pain must receive attention the underlying cause—non-compensation—must not be lost sight of. To treat this well in cases of aortic regurgitation requires much judgment and experience.

The drug treatment of angina pectoris in the interval between attacks.—Because the depression of blood pressure by the nitrites relieves the pain of angina, owing in great measure to their vaso-dilator effects, it is rational to believe that had we some agent which could for a longer period maintain such a vascular state a greater immunity from the recurrence of such attacks might be secured. Professor J. B. Bradbury of Cambridge reasoned thus when he gave his Bradshaw Lectures in 1895[23] That we may stave off an attack of angina by a timely dose of nitrite when a patient is about to perform some act likely by a rise of blood pressure to induce one may be admitted, but before regularly employing vaso-dilators which are less evanescent in their action than the nitrites we have to consider whether high arterial pressure as such is the cause of angina. That a habitually high-pressure pulse may be one of the causes of the atheroma and its consequences, of which angina may be the expression, can scarcely be doubted; but the actual cause of anginous attacks, the mine, as I have said, or local lesion once existent, seems to be less an equably high blood pressure than an irregular or intermittent one. To quote Heberden again, "Those who are attacked by this disease are wont to be seized with the most severe breast-pang while walking. ..... As soon, however, as the pain is arrested all distress is quieted in a moment (*totus angor momento conquiescit*)." Heberden's words perhaps convey the impression of a more rapid disappearance of pain on the cessation of those actions which induce them than always takes place, but they also establish the fact that it is the sudden or intermittent rise of blood pressure—it is, so to speak, the *blood stroke* rather than the blood pressure—which induces the pain, for the average blood pressure, even if it be high, is doubtless maintained in some such cases after the attack has passed off. When relief is observed in some cases of angina pectoris on the establishment of an apical bruit, indicating incompetency of the mitral valve, it is probably due not only to diminished blood pressure at the periphery, but also to lessened systolic impulse and perhaps to slightly diminished ventricular output. I am aware that in arguing thus I impugn in such cases the correctness of the theory of cardiac ischæmia. If, however, my argument be correct what we have to arrive at is securing an equable pressure, so far as possible, not necessarily a low pressure. The constant normal variation of pressure renders this object only partially attainable and this fact adds to the pity with which sufferers from angina must ever be regarded, for they carry as a condition of very life an enemy in their bosoms whose onslaught may be delivered at any moment and whose stroke cannot altogether be avoided even by the greatest watchfulness.

The higher nitrates, chiefly those of erythrol and mannitol, which Professor Bradbury recommends as more persistent vaso-dilators than the nitrites, have not come into general use, and it is perhaps questionable whether they are likely to do so. Sir Lauder Brunton's explanation of the *modus operandi* of the nitrite—namely, that they have a paretic influence upon vascular muscle—has been very generally adopted and Dr. Leech also satisfactorily showed in his Croonian lectures that the final, if not initial, action of these drugs upon the heart itself was of the same character. The nitrates, although not acting so powerfully as the nitrites, act in the same way and the continuous employment of a cardio vascular depressant is not calculated, on theoretical grounds, to be permanently beneficial. Dr. G. A. Gibson, however, writes:[24] "From the few observations which have been allowed me since the drug (erythrol tetra-nitrate) was brought forward no doubt has been left in my mind as to its value. Although somewhat less rapid in its effects it is much more persistent" (than the nitrites). Indeed, in some cases of failing heart with low tension and angina we are compelled circumspectly to use strophanthus. My own practice when this is necessary is to combine it with aromatic spirit of ammonia and spirit of nitrous ether. The ammonia, as Dr. Leech states,[25] retards the decomposition of the nitrous ether, and the nitrous ether tends to control the action of the cardiac tonic. This theory may be erroneous, but I have

[20] Op. cit., p. 71.
[21] Lectures on Disease of the Heart, third edition, London, 1898, p. 331.
[22] THE LANCET, July 15th, 1893, p. 124.
[23] THE LANCET, Nov. 16th, 1895, p. 1205.
[24] Diseases of the Heart and Aorta, p. 782.
[25] THE LANCET, July 22nd, 1893, p. 178.

found the combination at times to act well in practice. Digitalis given alone is frequently found to aggravate matters in sclerotic cases manifesting angina.

To secure, so far as possible, an equable blood pressure we must avoid as well as employ certain drugs. In this sense tobacco is a drug calculated to raise blood pressure and should theoretically be altogether avoided. The physician will, however, have to judge of the advisability of recommending this step by the character and circumstances of individual cases. To deprive a man long used to the solace of tobacco of his "smoke," if he be not a stoic, may be to precipitate rather than to obviate angina. But in any case permission to smoke must be inexorably limited to that minimum which will secure mental as well as vascular equability. We must also endeavour to remove indirect causes of vascular tension. We have been taught by physiologists the value of the play of neuro-vascular action in the splanchnic area as a safety-valve to threatened excess of vascular pressure elsewhere, and the use of certain agents which combat fermentation and promote the flow into the primæ viæ of the natural secretions which secure a normal blandness of intestinal content is of the first importance. Among such agents I should place in the first rank the milder mercurials—calomel and blue pill. I have cardio-vascular patients who have taken from half a grain to one grain of calomel once or twice a week regularly for a twelvemonth at a time with unquestionable benefit in maintaining an equable blood pressure. This practice on my part is no recent procedure but I did not derive it from my *alma mater*, the University of Edinburgh, for when I was in my professional infancy my clinical teacher, one of the ablest who ever felt a pulse—the late Professor Hughes Bennett—had, perhaps, among some dislikes one prime aversion—namely, mercury in every form. So that I began practice fully convinced that, did I prescribe that drug to my patients, I should shortly have around me a tremor-struck and gibbering public execrating me as the mercurialised prisoners in a quicksilver mine might their gaoler. From this delusion I was freed many years ago by the distinguished physician who is the President of this College and whose views on the beneficial effects of wisely administered mercurials in securing an equable blood pressure are now so well known. How mercurials act in these circumstances, whether as eliminants, as Sir William Broadbent suggests,[26] or as antiseptics, or as more direct vaso-dilators, it is not necessary for practical purposes to inquire. Their action is, without doubt, frequently beneficial in maintaining a certain softness and pliancy of pulse for long periods. General gastro-intestinal medication by alkalies or acids as required, or by the use of saline or other aperients, is indicated for the same reason—namely, to remove or to correct possible causes of intermittent blood pressure of an injurious character. It is possible that some of the good repute enjoyed by iodide of potassium in cardio-vascular sclerosis may be due to its efficacy in cases of specific origin, but such a supposition does not account for its general use by observant physicians. Although the drug had some vogue in such conditions before the advocacy by Dr. Balfour of its usefulness, there can be no doubt that his emphatic and authoritative utterances in its favour have done much to secure its general adoption as a cardio-vascular remedy or agent in treatment. He compares its action in one place to that of the nitrites[27] and in another refers its therapeutic effects in aortic aneurysm to the reduction by it of intra-arterial blood pressure.[28] The dose he recommends for continuous use as a vaso-dilator is two grains every 12 hours. The one objection to its employment has perhaps more weight in cases of angina pectoris than in cases not associated with these paroxysms—namely, its tendency at times to cause gastro-intestinal irritation and thus to provoke those reflexes which induce irregularities in vascular pressure. But the same objection applies to mercurials, and if the administration of both these useful agents be coupled with a scrupulous care as to the blandness and sufficiency of diet and the avoidance of condiments such as vinegar, ketchups, and effervescent wines and beverages, this objection is in no way comparable to the advantage which is constantly derived from their use in the intervals between attacks of angina. Concerning the *modus operandi* of the iodides in inducing a more equable vascular tone there is room for legitimate differences of opinion, for much obscurity attends our present knowledge of the subject. We must be content, meanwhile, to act empirically so long as our action is beneficial to the patient and wait for that enlightenment which science sooner or later affords those who industriously look for it and patiently expect its advent.

Drugs other than powerful analgesics and vaso-motor agents may be of service in some cases of angina. If the malady be associated with anæmia due to some specific cause such as malaria, or be attributable, as appears to be the case in some instances, to lead poisoning, or be aggravated by defective blood states of the kind more commonly met with, the treatment of the accidental state, *secundum artem*, by such agents as arsenic or iron or iodide of potassium, as the case may be, is calculated to be of benefit. In functional or inorganic cases they may be all that is necessary to effect a cure when coupled with attention to general hygiene.

What has been said as regards the treatment of angina pectoris applies in the main to the treatment of angina sine dolore or syncope trepidosa. As Dr. Leech has remarked, "although the ultimate influence of the nitrites on the cardiac muscle is depressant, it is probable that by inducing a larger number of smaller contractions the nitrites help to unburden an overladen heart, even if the individual contractions be less powerful than normal."[29] But in the syncopal variety of the complaint, if we still classify it with Heberden's disease, hypodermic injections of strychnine find appropriate and, at times, life-saving employment. M. Huchard has well remarked[30] that in some severe cases of painful angina the pain itself is the best safeguard against an overdose of a direct analgesic like morphia. So, also, it may be equally cogently maintained that in painless or syncopal angina the syncopal state is the best antidote against the injurious influence of a somewhat bold use of strychnine. With this powerful agent we may usefully combine, as in the case I have related, the tincture of strophanthus, which acts as powerfully on the heart as the preparations of digitalis and is less likely than the latter to raise a peripheral obstacle to the circulation as it acts less on the distant arterioles. Such a combination may be to syncopal angina what opium, chloroform, and atropine are to painful angina of a severe type.

We have now discussed with such fulness as the limits of our time will permit the nature, causes, and treatment of cardiac pain. Great as still is the obscurity of many important questions which are raised by a study of Heberden's disease, the retrospect of our progress in the knowledge of angina pectoris since he wrote in so masterly a manner is not discouraging. The ever-compelling future which justifies and renders interesting the present may be confidently predicted to show no lessening of the rate of progress which the last century has exhibited; and it does not require the vision of the seer to predict that much that is still a sealed book to us in the matters we have been considering will a century hence or sooner be common knowledge. But that this devoutly to be desired end may be reached thought must ever be the handmaid of observation and we must be careful to rear the superstructure of our conclusions on the rock of patiently verified fact.

[26] The Pulse, 1890, p. 182.
[27] Op. cit., p. 379.
[28] Op. cit., p. 467.

[29] Croonian Lectures, THE LANCET, July 24th, 1893, p. 150.
[30] Op. cit., p. 676.

HYSTERECTOMY FOR UTERINE FIBROID DISEASE IN EARLY PREGNANCY.

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THREE somewhat interesting cases of hysterectomy during early pregnancy have recently occurred in my own practice. In all there was a fibroid in the lower segment of the uterus posteriorly, preventing normal labour or delivery through the vagina by any means and in all immediate removal of the uterus seemed to be the best course to pursue in the interests of the patient.

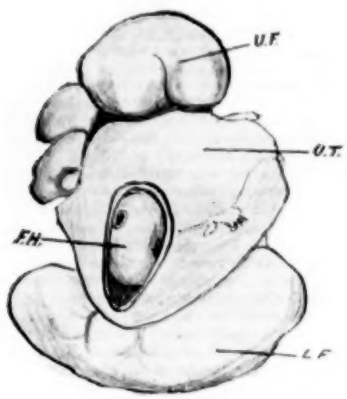
I need not discuss the important question of the treatment of the gravid fibroid uterus at length, as that has been already done by well-known writers, for instance by Dr. C. J.

Cullingworth,[1] who recently wrote an interesting article on the subject well worth perusal and by Mr. J. Bland-Sutton whose papers on the same question appeared in THE LANCET.[2] All obstetricians and surgeons who undertake hysterectomy should study Dr. Leopold Thumim's valuable monograph on operative experience since 1885.[3] There was an excellent discussion at a meeting of the Obstetrical Society of London in 1901 after the reading of a communication by Dr. Archibald Donald on fibroid tumours complicating pregnancy and labour, to which I shall have occasion to refer. I will now relate the three cases in my own recent experience. I am indebted to my friend and colleague, Dr. C. Hubert Roberts, for the instructive drawings illustrating the relation of the tumour to the uterine cavity.

CASE 1. *Pregnancy in an elderly primipara; uterus lying between two fibroid outgrowths, one obstructing the pelvis; hysterectomy at the fifth month; recovery.*—A full report of this case was published in THE LANCET.[4] The patient was 40 years old. She had been married two years and had never been pregnant till on this occasion. I watched the progress of the pregnancy for a month and found that a large fibroid mass behind and below the uterus did not rise, but, on the contrary, was beginning to block the pelvis. In consultation with Dr. Roberts it was agreed that it was not advisable to wait till term and then to undertake a Cæsarean section. I therefore performed retro-peritoneal hysterectomy in the fifth month of pregnancy on Nov. 1st, 1900. The patient was in good health in July, 1902.

The illustration (Fig. 1) is reduced from a drawing in the Transactions of the Obstetrical Society, vol. xliii., 1901,

FIG. 1.



Uterus and tumour, Case 1, seen from in front. U.T., Uterus. F. H., Fœtal head, showing one ear; three fingers are also visible. U. F., Upper fibroid which occupied the right hypochondrium and did not interfere with the pregnancy. L. F., Lower fibroid which blocked the pelvis. (Museum, St. Bartholomew's Hospital, Pathological Series XLIV., 5080, C.).

pl. 12. The upper tumour occupied the right hypochondrium; the lower, which sprang from the back of the posterior segment of the uterus, fitted closely into the pelvic cavity and pushed the cervix up against the pubes. Both fibroids were very hard and had the lower been pushed upwards the uterus might have been ruptured between them like a peach between two stones. The specimen is preserved in the museum of St. Bartholomew's Hospital.

CASE 2. *Fibroid uterus; numerous abortions; pregnancy to term, child saved; six years later pregnancy with large fibroid in the posterior part of the inferior segment involving the cervix; violent attacks of pain; hysterectomy at the fifth month; recovery.*—A woman, aged 39 years, who had been married 17 years, was brought to me by Mr. C. Eastwick-Field of Midhurst on Jan. 3rd, 1902, as he suspected pregnancy complicated by uterine fibroid. She had aborted no less than nine times, the last occasion being six years ago; then she became pregnant again and by careful treatment Mr. Eastwick-Field succeeded in keeping off the usual accident, so that she was safely delivered at term of a healthy boy, now five years old. Ten years ago she was under treatment in a hospital for dysmenorrhœa. Menstruation was regular till August 20th, 1901, when it was last seen. About a week earlier the patient noticed a small lump somewhat to the right of the middle line in the hypogastrium. This lump enlarged till the end of October and since then it seemed to remain stationary. In the second week of October there was discharge for several days and two large clots were passed. During November a watery, brownish, and odourless discharge escaped from the vagina for nearly a fortnight, then watery fluid came away in quantities; it ceased and for the last five weeks there was no more discharge. Morning sickness began at the end of September and lasted till the end of November, after which date it ceased altogether. On Dec. 28th the patient noticed a second lump in the left iliac fossa; she thought that she felt movements in it. On the same day violent labour-like pains set in and lasted for nearly three days. The patient had always experienced difficulty in holding urine long since the birth of her child and the trouble increased during the present pregnancy. Contractions were detected in the mass on the left, the vagina showed purplish colouration, and the os was very patulous. The mass on the right felt very hard; on that account the condition was suspected to be more probably fibroid complicating pregnancy than extra-uterine gestation. The patient was tall, anæmic, thin, and much exhausted from the recent violent attack of pain when she came under my care. I found an oval, hard, solid tumour occupying the hypogastrium and right iliac fossa; it reached to within three inches of the umbilicus and was fixed. On its left and behind it, separated by a distinct cleft, was an oval soft tumour reaching almost to the left hypochondrium and extending to the middle line. On palpation this soft tumour was felt to contract and a *souffle* was clearly audible. The cervix, wide and patulous, was completely displaced to the left but was not actually squeezed against the brim. The hard tumour on the right descended into the right fornix and indeed occupied all the pelvis except the corner to the left where the cervix lay, but did not come down low. It was continuous without any groove with the right side of the cervix; at that point the pulsations of the uterine artery were very clear. The whole was quite fixed. The breasts were small, the nipples were rather large, and a little moisture exuded from them on pressure. The urine was scanty and high-coloured; it contained a trace of albumin. The temperature never exceeded 99·2° F. during the four days before operation when she was under my observation. The pulse was rapid and exceeded 120 on Jan. 5th after a sleepless night due to attacks of pain round the tumour. There was no history of any general illness.

On consultation with Mr Eastwick-Field and Dr. Roberts it was agreed that owing to the position of the tumour it could not be pushed up. Three courses remained: (1) to empty the uterus; (2) to wait till term and to perform Cæsarean section; and (3) to remove the uterus at once. The first course, induction of abortion, might have been practicable, but even if so it would have entailed considerable danger and if successful would have saved for the patient a uterus which had already proved itself unfit to bear—a more than questionable advantage. Dr. Donald had recently reminded us[5] how, now that the results of operations on fibroid tumours have so much improved, induction of abortion is no longer the safest course for the patient. This obstetric operation in cases of pregnancy so seriously complicated with fibroid tumours as to render any interference necessary is a procedure not by any means free from danger. The cervix is more or less difficult to reach and the uterine cavity is distorted, hence retention of the placenta or the decidua and impediments to the free escape of blood and lochia are necessary results. The danger of primary septic infection is considerable, whilst the risk of a more chronic infection leading to degeneration

[1] Fibroid Tumours of the Uterus complicating Pregnancy, a Record of Personal Experience, St. Thomas's Hospital Reports, vol. xxviii.

[2] The Surgery of Pregnancy and Labour Complicated with Tumours, THE LANCET, Feb. 9th (p. 382), 16th (p. 452), and 23rd (p. 529), 1901.

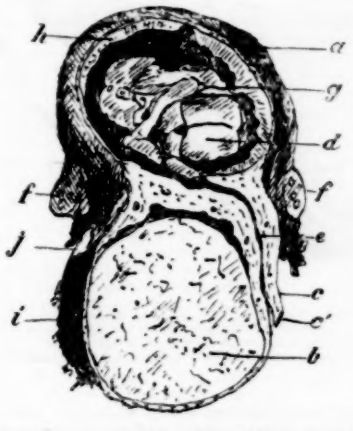
[3] Chirurgische Eingriffe bei Myomen der Gebärmutter in Schwangerschaft und Geburt, Archiv für Gynäkologie, vol. lxiv., p. 457.

[4] A Case of Hysterectomy for Uterine Fibroid in the Fifth Month of Pregnancy, THE LANCET, March 2nd, 1901, p. 621.

[5] Fibroid Tumours complicating Pregnancy and Labour, Transactions of the Obstetrical Society of London, vol. xliii., 1901, p. 180.

of the tumour or chronic septic changes in the appendages is yet greater. There is also serious danger of flooding, which is hard to control when a fibroid exists. Dr. Donald and others[6] referred to cases of induction of labour in fibroid disease of the uterus which had ended badly. Cæsarean section at term was contemplated but the patient's health had been failing for several weeks and was now very unsatisfactory. The pulse was high and dysuria was becoming severe. It therefore was questionable whether it would be safe to leave her alone till the middle of May on the chance of saving the child, which is usually ill-developed in cases where a large mass fixed in the pelvis interferes with the development of the uterine body. Immediate operation seemed to be the safest course. The advantages and dangers of all three procedures were carefully explained to the patient who unhesitatingly begged to be relieved at once from her miserable condition. On Jan. 6th another severe attack of pain came on with rapid pulse. I therefore operated on Jan. 7th, Mr. A. C. Butler-Smythe assisting. Mr. F. W. Collingwood administered gas and ether. The patient was placed in the Trendelenburg position. The abdominal incision had to be prolonged for one inch above the umbilicus. The bladder was not drawn up but lay in its usual position. There was no free blood or clot in the peritoneal cavity and there were no signs of old or recent peritonitis. The uterus and tumour were drawn out of the wound with ease. I found the ovaries lying high up on the mass; they looked unhealthy so I determined not to save them. The left broad ligament was tense. I tied the ovarian vessels and then the round ligament and divided the broad ligament. The right ovarian vessels, which were very large, were then secured. The right round ligament, which was greatly stretched as it ran over the face of the tumour, had pulled the peritoneum around the internal abdominal ring up against the tumour. This curious displacement, puzzling for the moment, was of course due to the manner in which the tumour had grown from behind and below to the right of the bladder upwards. I cut a flap of peritoneum on the front surface of the tumour

FIG. 2.



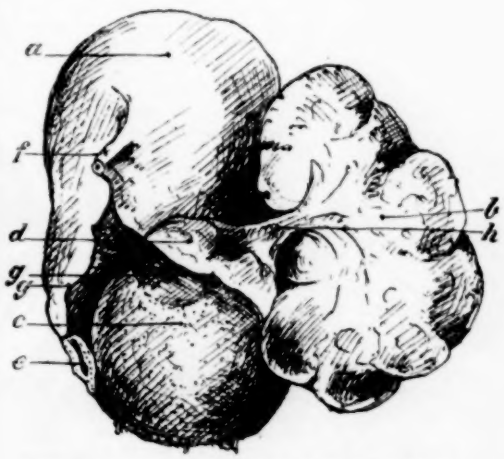
Uterus and tumour, Case 2, seen from in front. *a*, Body of uterus. *b*, Fibroid, mainly intracervical, which filled and blocked the pelvis and invaded the right broad ligament. *c*, Portion of cervix stretched over the fibroid and displaced to the left side of the pelvis. *c'*, Site of amputation through supravaginal portion of cervix. *d*, Fœtus. *e*, Canal of the cervix. *f*, *f*, Ovaries and tubes. *g*, Placenta and cord. *h*, Membranes. *i*, Portion of tumour (*b*) which was enucleated from the right broad ligament. *j*, Peritoneum, showing limits of enucleation.

which I then enucleated; it felt as though in a state of cystic degeneration. Then the uterine vessels were reached and secured. The flap was sewn over the stump after I had amputated the uterus, then the abdominal wound was closed. Recovery was uneventful and the patient was in good health in August, 1902. She had completely regained her strength.

[6] Loc. cit.

Dr. Cuthbert H. J. Lockyer took charge of the specimen prepared by Kaiserling's method. Its appearances are demonstrated in the illustration (Fig. 2) made by Dr. Roberts. The anterior part of the uterine wall has been removed, displaying the fœtus (a female) in the amniotic cavity, and the front part of the tumour has been cut away, showing its substance uniformly fibrous without any cysts. It is necrobiotic. It has been replaced in its capsule whence, as above explained, I had enucleated it during the operation. The fundus and upper part of the uterus are quite free from fibroid growths. The usual relations are very distorted, hence it is impossible to determine the precise site of origin of the tumour in the uterine walls, but the growth certainly arises in part from the lower segment and is also in part cervical. The supra-vaginal portion of the cervix, very thick, has been deflected out of the vertical line to the left. The

FIG. 3.



Uterus and tumour, Case 3, taken before the uterine cavity was opened to demonstrate the fœtus; view from in front with uterus slightly rotated to the right. *a*, Fundus of the uterus. *b*, Large, hard, pedunculated fibroid above the pelvis. *c*, *d*, Other fibroids, chiefly in the cervix. *e*, Cervix cut across at operation. *f*, Fallopian tube. *g*, *g*, Divided peritoneum, showing limits of enucleation. *h*, Pedicle of large fibroid (*b*). (Museum, Royal College of Surgeons of England.)

body of the uterus, softer than the tumour, was pressed to the left and thus compressed the cervix against the fibroid. It is clear that the fœtus was completely cut off from the pelvic cavity by the tumour.

CASE 3. *Nem[illegible] married woman, aged 30 years; hypogastric pains early in the first pregnancy; pedunculated fibroid in the abdomen and large fibroid in the lower segment posteriorly, involving the cervix and opening up the broad ligament; hysterectomy at the fourth month; recovery.*—A woman, aged 30 years, who had been married four months, was sent to me on March 22nd, 1902, by Mr. R. M. Boodle of Sittingbourne. She informed me that she had married in November, 1901, and the last period was seen on Dec. 13th to 17th. On Feb. 7th there was a slight show of blood. For two months the patient had suffered from pains in the hypogastrium. There was also much retching which continued through the day. She rightly believed that she was pregnant. Mr. Boodle detected a large tumour intimately connected with the uterus, whilst he found the os soft as in pregnancy. The patient was a robust young woman. The pulse was slow and strong, the temperature was normal, and the appetite was good. The vomiting and retching had increased since the pregnancy, but though her appetite had always been good milk or soups often made her sick. The breasts were fairly large; there was no oozing from the nipples on pressure. There was a hard, oval swelling in the middle of the abdomen, chiefly to the left, beginning a few inches above the pubes and extending almost to the left ribs. A soft body rose about three inches above the pubes, chiefly to the right.

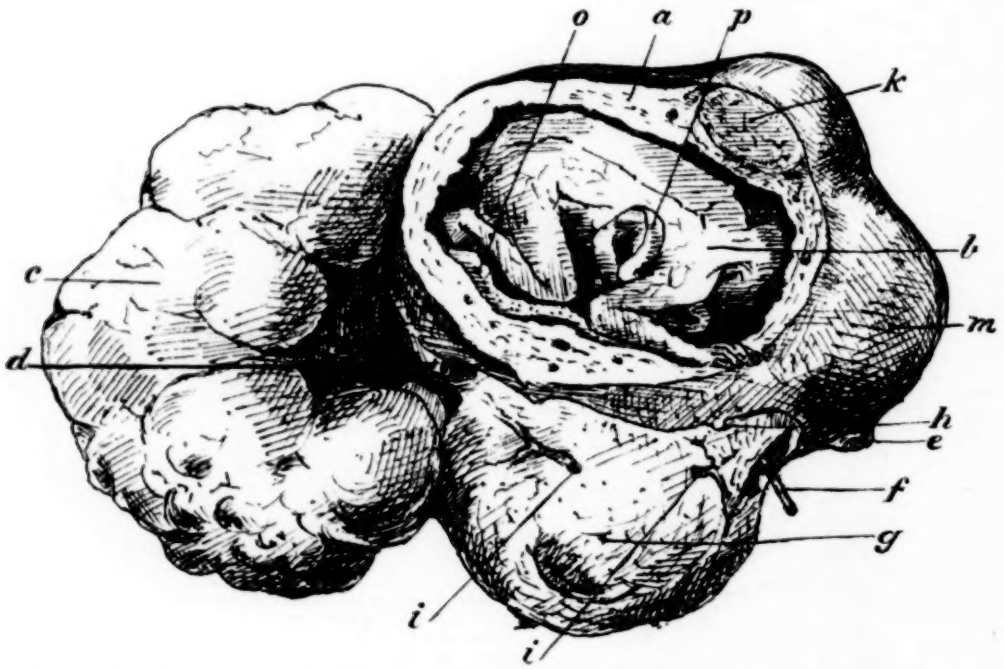
It was separated by a shallow groove from the hard swelling on the left above, but immediately to the left and on its own level lay a yet harder swelling, separated from the upper swelling by a deep groove. The upper hard swelling was freely moveable. The cervix was continuous with the lower hard mass on the left and with the soft mass. The os opened under the hard mass, part of which bulged in front of it and to the right as well as to the left. The masses were clearly united; the softest swelling—that is, the portion of the uterus including the fœtus—was cut off from the pelvis by the hardest swelling which above the pelvic brim lay entirely to the left of the uterine cavity. Dr. Roberts agreed with me, in consultation, that there was pregnancy with a fibroid uterus and that if left alone distension would result rendering delivery through the vagina impossible. There were two alternatives—hysterectomy at once or Cæsarean section at term. The matter was thoroughly explained to the patient and she dreaded waiting till term; it was clear, too, that the uterus was ill-fitted for pregnancy and likely to give trouble, even if it could be emptied of its contents. We decided, therefore, on hysterectomy and I determined to spare one or both ovaries if possible.

I operated on April 1st. The patient was kept in the Trendelenburg position throughout the operation. As I expected, the upper hard growth was a pedunculated subserous fibroid, the soft swelling was the uninvaded portion of the uterus, and the lower growth was a more sessile fibroid which was beginning to fill the pelvic brim. The lower growth had invaded the left broad ligament and the peritoneum of the vesico-uterine fold. I saved both ovaries, tied both round ligaments separately, divided the right broad ligament, and then made the anterior peritoneal flap. On turning it down I was able to enucleate the lower fibroid. A flap of peritoneum and muscular tissue was made posteriorly and then the uterus was amputated. The fœtal membranes were exposed but were not ruptured. The uterine arteries, which could only now be reached, spurted and were secured. Then I sewed the anterior peritoneal flap over the edge of the posterior flap, and in uniting by suture the layers of the broad ligament I turned in the cut surfaces of the tubes with the ligatures. Both ovaries were well developed. There was no shock and flatus passed freely at the end of 24 hours. Beef-tea enemata were given for two days as the patient had a strong distaste for barley-water or any fluid nourishment. On the fourth day the bowels were freely opened. There was no further trouble and convalescence was speedy. The patient was in good health in August, 1902.

Dr. Roberts took charge of the specimen, closing the cut surface of the uterus with a suture lest the organ should contract and expel its contents. The specimen was sent to the Museum of the Royal College of Surgeons of England and Mr. S. G. Shattock made of it an instructive preparation shown in the accompanying illustrations made by Dr. Roberts (Figs. 3 and 4). They show that the pedunculated growth was in no way obstructing the development of the uterus nor could it have interfered with labour, whilst the lower growth involved the cervix and posterior part of the inferior segment of the uterus. It extended into the left broad ligament; the limits of the peritoneum (Fig. 3, *g*, *g*, Fig. 4, *h*) are shown in the illustrations. It will be seen at a glance that the growth burrowed deeply into the pelvis and that it would have completely obstructed labour. The condition of the fœtus was remarkable. It was jammed between the big growth in the lower segment which had pushed its body up and a hard interstitial growth on the right which had distorted its head. Its subcutaneous veins showed much dilatation.

I have endeavoured to explain why immediate hysterectomy seemed to be the best course in all these cases. Myomectomy would have been impracticable. Pushing up the fibroid is a dangerous practice. A glance at Fig. 1 will demonstrate the peril of any attempt to press a hard, solid fibroid upwards when a soft gravid uterus lies above it. Such manipulations in the dark are unsurgical. We can never be absolutely certain that the pelvic tumour is a

FIG. 4.



Uterus and tumour, Case 5, seen from behind, with uterine cavity opened to display fœtus. *a*, Uterus showing several irregular fibroids in its walls (*k*, *m*). *b*, Fœtus, with head displaced by fibroid (*m*). *c*, Pedunculated hard fibroid springing from the left side of the uterus. *d*, Left Fallopian tube. *e*, Right Fallopian tube. *f*, Gibbs rod passed into the canal of the cervix. *g*, Large fibroid, enucleated part displayed. *h*, Divided peritoneum, showing limits of enucleation. *i*, *i*, Large vessels. *k*, *m*, Interstitial fibroids. *o*, Edge of placenta. *p*, Umbilical cord.

fibroid. Even if there be myomata in the uterus above the pelvis we must not forget that a hard ovarian tumour may develop and block the pelvis, but a tense ovarian cyst pressed down in the pelvis by a gravid uterus may feel very hard, as in a case which I reported in THE LANCET last spring.[7] The dangers of pressing on a cyst are evident. Franta reports cases where the pelvic tumour obstructing pregnancy or labour proved to be a hydatid.[8] As bimanual palpation of the pelvic mass is impossible when there is a gravid uterine cavity in front of it we can never be sure what we are attempting to push out of the pelvis.

A CASE OF FULMINATING APPENDICITIS WITH GENERAL SEPTIC PERITONITIS; OPERATION; RECOVERY.

BY J. HOWELL EVANS, M.A., M.B., M.CH. OXON., F.R.C.S. ENG.,

SENIOR DEMONSTRATOR OF ANATOMY AT ST. GEORGE'S HOSPITAL.

THE following case appears to deserve recording both on the grounds of its intrinsic interest and on account of the excellent illustration which it affords of certain main guiding principles which are essential to the successful treatment of a peritoneal infection. It also affords an opportunity of putting forward certain factors associated with this lesion which do not appear to have received very much attention hitherto.

A well developed, markedly intelligent, and extremely well cared for child, aged four years, the son of a medical man, previously in good health, complained of pain in his right side on the afternoon of Sept. 25th, 1902. Though the pain was present the child's condition was but little altered until, after midnight of the 26th, he vomited. A neighbouring practitioner finding little localised pain and no tumefaction or rigidity of the abdominal wall, associated his condition with a gastro-enteritis rather than with any appendicular lesion. Restlessness, vomiting, thirst, and a desire for the local application of ice to cool the lower part of the right side of the abdomen continued. Dr. F. W. Burton-Fanning saw the child at noon on the 27th. By this time I had been telegraphed for, and finding the pain somewhat localised, with slight rigidity over the right inguinal region accompanied by considerable febrile disturbances, advised certain necessary preparations for a probable operation. A few hours later, when I saw the patient with Dr. Burton-Fanning, the condition was briefly thus. The child's face was flushed and the aspect distinctly that of acute subdiaphragmatic serous inflammation, the twitching upper lip being drawn up in such a way as to show the teeth, with an expression of anxiety and nervous unrest accompanied with precautionary expiration. The temperature was 103·4° F. and the pulse was 120. Intelligence was quite clear and the pain was localised in the right inguinal region, where tenderness corresponded to the point of the pain. There was rigidity of the lowest segment of the right rectus abdominis muscle, but no localised tumour or dulness. Per rectum considerably more resistance was felt in the right upper portion of the pelvis.

An operation was performed on Sept. 27th. An incision was made in the usual manner, but at a slightly lower level and was carried through the superficial structures and obliquus externus. The other muscles of the abdominal wall were divided or separated in a plane at right angles to the superficial structures. Upon opening the peritoneal cavity a quantity of very offensive opaque fluid escaped. The vermiform appendix was found, anchored by its mesentery (mesenteriolum), to be floating in this fluid which welled up from the pelvic and perinephric regions. The mesenteriolum was ligatured so that the vessels contained within its layers were definitely secured and the appendix was removed distal to a single constricting basal ligature. After very gentle (from six to eight inches pressure) but copious irrigation had been carried out the cavity was gently swabbed out and the condition was further investigated. Adhesions are invariably formed, though occasionally to a very slight degree; so in this case the absence of any tendency of the intestines to bulge into the wound was taken as clinical evidence of this variety of nature's self-protecting endeavours. Accordingly three drainage-tubes of large calibre were introduced respectively down into the pelvis, up towards the right kidney, and on to the sacro-iliac synchondrosis, and in their tracks were inserted gauze drains. The wound was then partly closed and secondary sutures were passed in readiness. The usual absorbent dressings were applied and a rectal tube was introduced. The child had by this time recovered from anæsthesia and shortly afterwards passed some flatus.

Subsequently to the operation, from the first to the tenth hour, copious drinks of warm water, barley water, and diluted whey were given. Vomiting occurred at intervals and diuresis became established. A saline enema (eight ounces) was given but shortly returned and was followed in an hour by three small but very hard scybalous masses. The child dozed, waking to vomit or to relieve his thirst at longer intervals. From the tenth to the twentieth hour the facial aspect described above persisted, the abdomen was resistant all over, and the pulse continued almost imperceptible. Another enema (soap and water) succeeded in carrying away an extremely offensive collection of pale-coloured fæces. An intra-muscular injection was given consisting of one-sixteenth of a grain of acetate of morphia, one minim of tincture of digitalis, two minims of solution of strychnia, and water to one drachm. The child slept for an hour and appeared to be temporarily improved, but there still remained the unfavourable signs of restlessness, facial aspect, and an imperceptible pulse at the wrist. Food was now administered in the form of three ounces of sugar, half a drachm of salt, two ounces of gelatin, and water to one pint, five ounces being given at a time. About 20 hours after the operation the child, whose condition was growing worse, vomited about one and a quarter pints of black coffee-ground vomit—usually a sign premonitory of an impending fatal issue. Hereupon the dressings were removed, the drainage-tubes were taken out, and the wound was opened in its entirety; all adhesions were thoroughly broken down and the whole abdominal cavity was copiously irrigated by a contrivance which was extemporised from rubber tubing and which directed all fluid towards the wound. Large sheet-like gauze drains were placed in the direction of the liver and of the left kidney and to the bottom of the recto-vesical pouch. An intra-muscular injection was administered, consisting of one-sixteenth of a grain of acetate of morphia, one minim of tincture of digitalis, two minims of solution of strychnia, and water to one drachm. The large gauze drains were replaced after three hours by tubes with small intertubular gauze drains and at intervals of from three to four hours the child received a nutrient enema as follows: four ounces of sugar, one drachm of saline solution, four ounces of gelatin, and water to one pint, a half pint being given every four hours. A hypodermic injection consisting of half a minim of tincture of digitalis and one minim of solution of strychnia was also administered every two hours. For the greater part of the next ten hours the child slept, waking at intervals, when his thirst was quenched, and from this time onwards he progressed, slowly at first, but subsequently rapidly, to recovery.

An examination of the appendix by the naked eye showed it to be thickened throughout and to be four inches long. Its distal end was very much enlarged; it was perforated and contained a mass a little larger than a cherry-stone on the proximal side of which a fig seed was found. Microscopically all its coats were thickened and in the intercellular spaces numerous undifferentiated micro-organisms were to be seen. There was also thrombosis of the small veins. The enterolith consisted chiefly of epithelial cells, mucus, and micro-organisms.

The matters calling for explanation here are (1) the obstruction by the fig seed; (2) the formation of the enterolith; (3) the cause of the perforation and fulmination; (4) the thrombosis of the vessels; and (5) the scybala which were passed per rectum. 1. As to the obstruction, eight days prior to the attack the child had eaten some stewed figs from which the seed which was found had evidently come and this produced a certain amount of obstruction to the exit of the natural secretion of the epithelium. 2. This irritation, associated with micro-organisms, produced inflammatory changes which in the earlier stage of hyperæmia

[7] Ovarian Tumours and Ovariotomy during and after Pregnancy, THE LANCET, Feb. 8th, 1902, p. 356.

[8] Kystes Hydatiques pendant la Grossesse, l'Accouchement et les Suites de Couches, Annales de Gynécologie et d'Obstétrique, March, 1902.

brought about the increased secretory activity and multiplication of the epithelial cells, resulting in the formation of the enterolith. Every portion of the intestines may be considered to form its own fæces, as shown by (*a*) the meconium in the newly-born, (*b*) the experiments of Thiry and Vella, and (*c*) clinical observations in cases of colotomy. 3 and 4. The irritants, increasing the morbid process, led to an extensive involvement of all the submucous, intramuscular and perivascular subserous lymphatic channels, culminating in an acute endophlebitis and consequent gangrene, which was more marked at the point furthest removed from the mesenteriolum where the perforation—probably accelerated by the pressure of the enterolith—was found. 5 The scybala which are invariably passed by those patients who recover from acute appendicitis a few days after the attack have been considered by some eminent authorities as the "offending particles"; but in the writer's opinion they are nothing more than the usual fæces deprived of their fluidity while in the sigmoid flexure. The determining factors in a fulminating appendicitis are similar to those of an acute cellulitis elsewhere. They are: (1) the virulence of the micro-organisms, and (2) the degree of venous thrombosis leading to gangrene.

With regard to the treatment of this case I aimed originally at the following objects: (1) removal of the focus; (2) washing away all poison as yet unabsorbed; and (3) promotion of excretion of the poisons absorbed by the natural means—viz., the kidneys, the bowels, the stomach, and the skin—and not until these important organs were acting satisfactorily was any drug administered which could diminish excretion. It would have been better, in all probability, to have established a long-continued subcutaneous saline infusion, as carried out systematically by Kocher of Berne, or saline and sugar, so as to induce an efficient diuresis. In regard to feeding the patient the most essential and easily assimilable articles with abundance of fluid were given.

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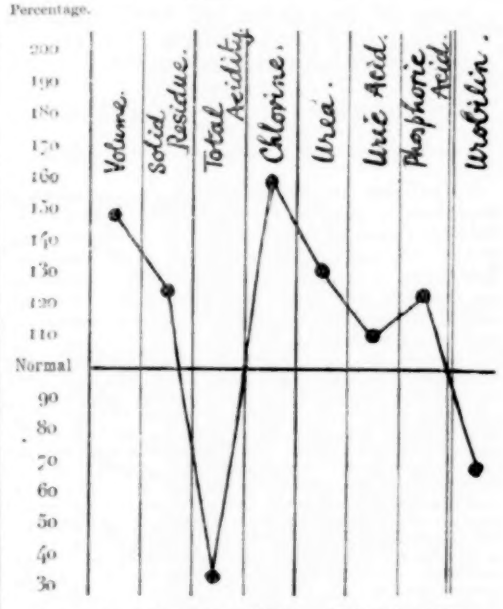
THE LOW PHOSPHATES AND UREA IN THE URINE OF THE TUBERCULOUS.

BY A. W. GILCHRIST, B.Sc., M.D. PARIS, M.R.C.S. ENG.

IN a paper on the Use of Urinary Graphs, written on the occasion of the last meeting of the British Medical Association, and which I shortly hope to publish, I referred to the custom gradually prevailing in France of having frequent recourse for diagnostic purposes to thorough examinations of the urine and advocated the adoption of the method[1] of representing the main features of the analytical results in the form of a semeiological *graphique* or chart. For this purpose I selected for description from my notes a copy of what is usually known as a complete analysis made by M. Richard, our biological chemist at Nice, and prepared a series of charts illustrative of the character of the urine in the commoner forms of disease. It is one of this series, showing the typical nutritive rhythm,[2] so to express it, of the early tuberculous state, that is given below. It would be difficult, perhaps, to find a more favourable opportunity than that which presents itself at present to draw attention to the subject in question. The presumably growing importance of this branch, though only a minor one, of a highly progressive science, may be gathered from the general tone of Dr. W. D. Halliburton's recently published address on the Present Position of Chemical Physiology and the leading article of THE LANCET[3] that accompanies it. But a further inducement arises from the appearance at the same time of a paper by Mr. E. G. Clayton[4] on Some Observed Variations in the Phosphates and Urea of Urine, and a letter on the Low Phosphates and Urea in the Urine of the Tuberculous from Dr. Harper.[5] I will, therefore, venture upon a few remarks suggested by the use of the analyses and the charts I have mentioned and refer at the same time to some of the important questions raised by Mr. Clayton's and Dr. Harper's papers.

It would seem to me that the first conclusion that will at once be arrived at on comparing the information derived from M. Richard's chart and the analytical deductions drawn from similar observations by the writers to whom allusion has just been made, will be to confirm the now generally recognised fact that an analysis of urine, except in some very obvious cases, is of very doubtful value unless the specimen examined is part of the collective fluid excreted during a complete cycle of 24 hours.[6] If this



The character of the graph is slightly modified by the action, probably, of arsenic which was given for some days previously to the analysis being made. In an untreated case of common febrile—that is, not torpid—phthisis the phosphates would usually be higher than the fixed elements.

essential condition had been observed Dr. Harper, for instance, would never probably have ventured upon the statement he makes in the paper to which I refer. He would, on the contrary, have recognised that one of the salient characteristics of the urine in tuberculosis is to furnish, not a deficiency, but a marked increase in the total solids. This fact is evident in column 2 of the appended graph, while a reference to the high volume indicated in column 1 will show that although single specimens of urine, such as Dr. Harper seems to have habitually examined, may be small in quantity and of low specific gravity, the daily output of the renal excretion and the fixed elements it contains may both be excessive. The frequent use of a graph would also lead to the remark that this increased volume of urine is invariable in the incipient stages of ordinary phthisis and that the feeble tenure in urobilin[7] as shown in the last column, and which possibly confirmed Dr. Harper in his false impression, is another constant feature in this phase of the evolution of pulmonary tubercle. Column 3 points to the low acidity, 30 per cent., as compared with the adopted varying normal of 100 per cent.[8] of the

[1] Particularly that of Dr. Gautrelet, to whom the system is originally due.

[2] Avec une bonne analyse d'urine, on peut lire en quelque sorte dans la nutrition et dans ses actes si multiples et savoir comment le malade s'alimente, assimile et désassimile. On peut mesurer non seulement les actes généraux des échanges organiques, mais encore les activités particulières de la plupart des organes (Professor Robin).

[3] THE LANCET, Sept. 20th, 1902, pp. 787 and 820.

[4] THE LANCET, Sept. 6th, 1902, p. 686.

[5] THE LANCET, Sept. 13th, 1902, p. 776.

[6] Ogden's Clinical Examination of the Urine.

[7] Urine pale on emission—here the remark is general—may contain enough of the chromogens to deepen in colour when exposure to sunlight converts these substances into true pigments (MacMunn and Gautrelet).

[8] The factors involved are the age and height of the person concerned, and sometimes the chest measurement, with the mean between the actual and theoretical weight; it is obviously illusory to expect accurate results from an average for comparison of 1500 cubic centimetres, which makes no special exceptions for age, weight, sex, constitution, diet, or, above all, for medicinal treatment, as, for instance, by alkalies.

subjects of what might be called the "consumptive" diathesis, that is, of those predisposed to tuberculosis in particular and to infection or contagion in general. The urea is high, 132 per cent. in column 5, as well as the phosphates, 126 per cent. in column 7, both additional features of phthisis in all but the cachectic stages, unless it be a question of local disease as in Mr. Clayton's case of tubercle of the bladder, or of early forms of chronic fibrous pneumonia or tuberculous cirrhosis of the lung evolving in a gouty and hence unyielding organic environment.

A last reference to M. Richard's chart will show the chlorides reaching the high percentage of 160, a fact implying that Mr. Clayton has not been any more fortunate in this respect than in the other material for reference which he has provided for Dr. Harper. His statement, in fact, that "the salt does not present such notable departures from normal proportions" is also erroneous. Calculating by densimetry, since the extract is not given—that is, multiplying the last figures of the specific gravity by 2·225 to obtain 25 59 grammes—the total solids per litre in the case of tubercle of the bladder—as one term, and converting into chlorine the ascertained amount of chlorides (8·19 grammes) for the second, there is furnished a ratio of about 20 per cent. instead of 10 per cent., the figure obtained when the total solids and the chlorides of normal urine are compared.

If Mr. Clayton had given the salt in samples A, B, and C, it would probably have shown a relative deficiency almost as much below the normal as the excess is great in his first case. In fact, this inverse proportion in the excretion of chlorine in the two diseases between which he seeks to establish an analogy is commonly employed by French writers on urology and notably by Professor Robin, M. Armand Gautier, and Dr. Gautrelet, for the diametrically opposite purpose of illustrating one of the chief contrasting features of these two respective conditions. The subjects of tuberculosis, we have already seen, belong to the "hypo-acid" group, where the chlorine progresses with the polyuria, while those liable to arthritic or gouty glycosuria (the term of "diabetes mellitus" is scarcely justifiable with so small an amount of sugar as 1·23 grammes per litre) excrete in the early stages, unless the high arterial tension of supervening arterio-sclerosis affects the volume, small quantities of salt in otherwise abnormally concentrated and[9] hyperacid urine (Specimen B, to which attention was drawn, has a density of 1028, and Specimen A is described as "very acid"). Not less fallacious is Mr. Clayton's remark regarding the excretion of phosphoric acid; the amount of this constituent for such a case as he mentions would be, in the absence of marked congestion of the liver, about 1·33 grammes, whereas the average of the quantities he finds is 1·67 grammes—that is, an excess of over 12 per cent. above the usual pathological normal—another indication, I may add, of mild glycosuria readily yielding to treatment by a non-alcoholic and light nitrogenous and vegetable diet. Had the investigation of Specimens A, B, and C been carried further there would doubtless have been found amongst the epithelium débris and mucous deposit a certain number of renal cells pointing to uratic or oxalic irritation, an appreciable quantity of peptones[10] and biliary acids,[11] an excess of urobilin[12] indicative of insufficient oxidation and consequent aberrant hepatic assimilation; and what is of greater importance than quantitating the sulphuric acid[13] the occurrence could presumably have been shown of the ethereal sulphates indican and skatol (indoxyl- and skatoxyl-sulphate of potassium) which point to auto-intoxication from abnormal fermentations and albuminoid putrefactions, more especially in the intestinal tract.

I am sorry not to have access at present to Dr. Harper's papers on Urea. I entirely agree, as will be gathered from the preceding remarks, with his opinion that the less animal or nitrogenous food ingested the greater the incidence of tuberculosis, though I question the utility of urea to supply the nitrogen which we have seen to be consumed in excess in phthisis. Theoretically the exhibition of a substance like urea, which besides being a completely oxidised body is now generally regarded as an active physiological diuretic, is scarcely suited to a condition where, in the early stages at least, polyuria and demineralisation of the tissues are predominant, while practically I am not certain that its use has given permanently good results at Leysin, where the method has been proposed. It would be more scientific, or more in keeping with successful experiment, to follow Professor Richet and M. Héricourt in administering the cold extract of raw meat which probably acts in virtue of a special ferment,[14] and to trust to a highly nitrogenous and fatty diet, which raises the general acidity of the system, arrests excessive disassimilation, and diminishes the organic exchanges.

In this respect the recent investigations of Professor Robin and M. Maurice Binet into the chemistry of respiratory metabolism are highly instructive. These writers have shown that, contrary to the generally received opinion, but quite in conformity with the leading idea pervading this paper, the subjects of tuberculosis, whether in the hereditary protopathic or infective stages, absorb an excessive quantity of oxygen, produce a correspondingly large amount of carbonic acid, and fix in their tissues an abnormally high proportion of oxygen.[15] Thus, to mention only one of the many results of numerous experiments, it was found that while the antagonistic state of "arthritisme" gave a consumption of oxygen per minute kilogramme of 4·41 cubic centimetres and the normal adult 5·13 cubic centimetres, the amount in the tuberculous subject attained the significant figure of 9·45 cubic centimetres. Such patients, then, are "consumptives" in the true Hippocratic and English sense of the word. The treatment of their condition should not therefore consist in having recourse to tonics and nutritive stimulants, but rather in supplying, as Professor Robin suggests, such foods and medicines as are restrictive in their action or that may attract to themselves part of the disintegrating chemical agent which is consuming the body. It is on the nature of the soil,[16] indeed, as Dr. Dickinson has lately said, that our attention should be centred in the treatment and, it should be added, the prevention of tuberculosis.

Nice.

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[9] Herter (Lectures on Chemical Pathology, p. 32) says: "It is convenient to use the term of *acidosis* to designate the slighter grades of acid intoxication."

[10] Herter (loc. cit., p. 81) observes, presumably on the authority of Kutscher: "But it has been almost conclusively shown that peptones do not occur in the urine in pathological states and that the substances formerly described as such are in reality albumoses. Gautrelet, however, contends that peptonuria is a constant and reliable sign of hepatic insufficiency and that if observers such as Hofmeister, Rohmann, Schmidt, and Mulheim, have failed to find these bodies in the portal blood during digestion, and have attributed their disappearance to leucocytic action in the intestinal mucous membrane, it is that the methods of investigation were defective. This defect extends to the usual biuret test which fails to show a reaction in solutions containing less than 1 per cent. of peptones" (Constance et valeur sémélologique de la peptonurie dans les Maladies par Aberration de la Nutrition, Paris, 1902).

[11] The excretion of these acids may mean a certain degree of catarrhal biliary retention. When Hay's or Gmelin's reactions fail to detect their presence, recourse, it has recently been stated, may be had to a very sensitive method founded upon the superficial tension of fluids. (Recherches sur la Tension Superficielle des Urines, Billard et Prof. F. Malby, Centre Médical, July, 1902.)

[12] The tenure in urobilin varies with sufficient constancy to be a fairly reliable guide, inversely as the coefficient of oxidation (*rapport azoturique*), a figure obtained by taking the ratio between the total nitrogen of the urine and the nitrogen excreted as urea. This quotient, like those in the appended graph, is expressed as a percentage. The generally accepted normal is 84 per cent. (Bouchard, Robin); it is usually higher in tuberculosis and lower than this figure in cases of retarded nutrition.

[13] Most of the information derived from determining the total sulphates may be inferred from the coefficient of oxidation. In some cases, however, it is useful to compare the total sulphuric acid with the incompletely oxidised sulphur. Robin calls this ratio the coefficient of hepatic activity and adds: "When the decrease of this coefficient coincides with the presence of urobilin or of uroerythrin its value is all the more appreciable."

[14] Hypotheses, proverbially fragile, are little better than aids to memory in seeking to explain whether this muscular serum, as it is called, and which is found to be a specific in inoculated tuberculosis, acts as an antitoxin, a stimulant to phagocytosis, or simply as a special tonic to the nervous system intrusted with the defence of the organism. Professor Richet, however, in a suggestion ("comme moyen mnémotechnique") which seems to be prompted by Ehrlich's side-chain theory, supposes that the serum in question may act by a kind of nutritive substitution, that is, this liquid may contain a substance which, impregnating the nerve cells that preside over nutrition, thus prevents their invasion and consequent inhibition by the toxins of tuberculosis.

[15] Or hydroxyl ions, as the modern bio-chemist might perhaps prefer to express it. (See Dr. Herter, loc. cit.)

[16] It is remarkable that in scrofula, "ce terrain ou germent surtout les tuberculoses locales," respiratory metabolism is not exaggerated (Robin, Bulletin de l'Académie de Médecine, January, 1902), a fact in keeping with the habitual type of strumous urinary excretion, which though conforming in general to the "hypo-acid" group, is said to differ from that of the subjects of pre-tuberculous conditions in a lower output of urea and a varying relative excess of uric acid and the earthy phosphates. Comby admits two categories of the scrofulous and uses in their designation terms analogous to those we apply to gout—the florid and atonic varieties. It will be seen—and this remark is *à propos* of a recent discussion in THE LANCET—that the best French authorities display no reluctance in retaining the debated but convenient term of "scrofula."

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

SARCOMA OF THE KIDNEY IN A CHILD THREE YEARS AND TEN MONTHS OLD; NEPHRECTOMY; RECOVERY.

BY THOMAS JAMES WALKER, M.D LOND., M.R.C.S. ENG.,
SENIOR SURGEON TO THE PETERBOROUGH INFIRMARY.

APROPOS of the case of sarcoma of the kidney in a child, aged six years, reported by Dr J. Eustace Webb in THE LANCET of Oct. 18th, p. 1043, I think the following condensed notes of a case of sarcoma of the kidney in a child successfully operated upon are worthy of record.

A female child, aged three years and ten months, was taken to Dr. C. N. Elliott of Oundle on Sept. 26th, suffering from pain in the abdomen, diarrhœa, and inability to pass urine. Dr. Elliott found the bladder distended, a high temperature (103·8° F.), and pain in the left iliac fossa. The child was sent home and put to bed and a trained nurse was engaged. The catheter was required for four days, when frequent micturition set in. The temperature ran up each evening to 103°. There were some pain and distinct swelling in the upper part of the right iliac fossa. A provisional diagnosis of appendicitis was made.

On Oct. 10th I saw the child with Dr. Elliott. There were slight distension of the bowels and a tumour in the abdomen at the junction of the lumbar and iliac region, the most prominent part being above the line from the umbilicus to the anterior spine of the ilium. The chart showed an evening temperature of close upon 104°, with an occasional drop for 24 hours. I could not make out the tumour in the lumbar region behind and I concluded that it was inflammatory.

The patient remained much in the same state up to my second visit a week later. The tumour then was more prominent. Percussion did not much assist the diagnosis and I determined to explore. I made an incision and opened the peritoneum sufficiently to introduce the finger. I found the tumour retro-peritoneal, extending from beneath the liver into the iliac fossa and across the spine beyond the middle line. Being satisfied that the growth was renal I closed the peritoneal wound, made a T-shaped extension of the incision, and with considerable difficulty enucleated and removed the kidney. The child bore the operation well and made an uninterrupted recovery, the temperature falling after the operation and remaining normal or subnormal. The kidney was infiltrated with sarcoma; it was of the size of my half-closed hand.

As to the ultimate result of such a case, unfortunately, the prognosis must be most unfavourable.

Peterborough.

A CASE OF SCARLET FEVER FOLLOWED BY AN INTERESTING RELAPSE.

BY HENRY FRASER, M.D. ABERD., D.P.H.,
RESIDENT MEDICAL OFFICER, ABERDEEN FEVER HOSPITAL.

THE occurrence of relapses in scarlet fever has not infrequently been recorded. Many hold that in the majority of such cases the primary attack has not been scarlet fever and that the relapse is merely the result of an infection by confinement in a scarlet fever ward. Hence the importance of recording such cases as the present one, where no apparent doubt can attach itself to the diagnosis either of the primary affection or of that which evinced itself in the relapse.

The patient was aged 20 years and was admitted into the Aberdeen Fever Hospital on June 6th, 1902, suffering from scarlet fever. There was a well-marked punctate rash accompanied by a suffused erythematous blush extending from the neck downwards over all the body. The eruption had appeared first on the day previous to admission. The tongue was much reddened and papillated; it was a typical "raspberry" tongue. The throat was much inflamed but there was no membranous deposit. The submaxillary glands were slightly enlarged. There was no desquamation and the urine was normal. The morning temperature was 99° F. and that in the evening 99·6°. On the day after admission the patient's temperature rose to 102·6° and on the next day it was 103°. Rheumatic pains in the left shoulder and both knee-joints were now complained of. Salicin was administered. On the 9th a carbuncle developed on the nape of the neck. Signs of fluctuation were evident on the 12th, when an incision was made and the purulent contents were evacuated. Healing took place rapidly. The rheumatism disappeared, the temperature became normal, and on the 19th the patient was able to get up. On the 12th desquamation had been observed on the neck and hands; it gradually increased in amount and by the 22nd it was very profuse. On the 25th—that is, six days after the patient had been allowed to get up—a severe attack of diarrhœa and vomiting occurred and on the same evening a very pronounced punctate rash appeared which by the next morning extended over the whole body, the face being free. The tongue was thickly coated, the throat was much swollen and congested, and the submaxillary glands were enlarged. The temperature had risen from normal to 102·6°. The urine contained no albumin. By the 28th—that is, three days later—the rash was fading, the tongue was papillated and cleaning, and there was some greyish membranous deposit on the throat, but the diphtheria bacillus was not found. The temperature gradually fell to normal, remaining so until July 3rd, when there was a slight rise for a day or two and the urine contained a small amount of albumin. Soon afterwards the patient became convalescent. As already mentioned, the desquamation which followed the first attack was extremely abundant; that following the relapse commenced and extended in an identical manner. On the hands it was observed as a powdery substance and was easily seen when drying the hands after washing. On the feet and legs it was, save for the lessened amount, similar in every respect to the primary desquamation. On August 4th, after 60 days in hospital, the patient was discharged free from infection.

As regards the origin of the relapse or second attack two explanations are, I think, possible: firstly, that it was simply an instance of auto-intoxication in a patient whose immunity was short-lived; secondly, that a fresh strain of the poison was contracted by the patient on getting up. Coupled with the fact that immunity after the first attack was not fully developed, the latter theory is, I think, the more probable. One not infrequently sees cases in hospital where a doubt exists as to the diagnosis of scarlet fever and such not infrequently contract scarlet fever, but in the vast majority of cases this occurs only when the patient is allowed to be up and mixes with the other patients in the ward; especially is this noticeable in the case of children.

Aberdeen.

A CURIOUS CASE OF ACUTE LEAD POISONING IN AN INFANT.

BY HAROLD C. CADMAN, M.B.

ON Nov. 8th, 1902, I was called to see an infant, five weeks old, who had been very constipated for some days and for some time had been gradually losing flesh; he had had several attacks of colic and vomited curdled milk. When an infant appears to suffer from abdominal pain I always make a point of looking to the state of the feeding-bottle, if one is used, but in this case the infant was suckled by the mother who was a healthy-looking woman. The abdomen of the child was hard and swollen and the umbilicus was retracted but not the abdomen itself. The child's general appearance was unusual. He looked too ill to be suffering from ordinary stomach-ache, and his face had such a peculiar grey colour that I thought he might be actually dying then. Nothing to indicate obstruction could be felt in the abdomen. I ordered hot flannels and gave three powders consisting of an eighth of a grain of compound powder of ipecacuanha and two and a half grains of carbonate of bismuth, a teaspoonful of castor oil to be taken previously. I also directed the mother to give up nursing for 24 hours and to give the infant barley water with a spoon. On the 9th the bowels had been moved; the child had been fairly free from pain and had only taken one

powder as these were only to be given if pain came on again. The barley water was directed to be continued and no breast milk to be given. On the 10th the child looked rather better; the abdomen was not so hard, he had had another attack of colic but not so severe, and had taken another powder. He had vomited. I said that the mother might now give him the breast. On her preparing to do this I saw that she had a piece of lead over the nipple, and on her taking this off I saw that the milk had dissolved some of the lead; the skin of the nipple was sodden and white, looking like skin after lead lotion had been used. It now became evident that the infant was suffering from lead poisoning. The advertisement on the box which contained the shields runs as follows:—"Dr. ——'s Metallic Nipple Shields.—The nipple is immersed in a solution of lactate of lead. The whole contained in a reservoir, as it were in the cavity of the shield which prevents pressure."

A napkin saturated with the infant's urine only was sent to me on Nov. 11th. I cut it into pieces and took one-fourth of the whole to Mr. W. H Gregg of Church-street, Barnoldswick, to whom I am much indebted for the trouble and thought which he gave to the analysis. He boiled the napkin in distilled water and found that the resulting liquid when evaporated to a small bulk and treated with potassium bichromate yielded a distinct precipitate of yellow chromate of lead. Another portion of the same liquid treated with potassium iodide yielded distinct crystals of iodide of lead in golden yellow flat square plates. A piece of the napkin placed in sulphuretted hydrogen showed several black specks of sulphide of lead. I still have more than half the napkin for examination if necessary. The child is still mending (Nov. 26th).

Barnoldswick.

DISLOCATION OF THE HIP-JOINT IN A CHILD.

BY WILLIAM MACDONALD, M.B., C.M. ABERD., L.R.C.P., L.R.C.S. EDIN., L.F.P.S. GLASG.

As dislocations are of comparatively rare occurrence in young children the following case of simple traumatic dislocation of the hip-joint may be worth while recording.

On August 16th, 1901, a female child, aged two years and eight months, was brought to me by her mother who stated that the patient had fallen on that morning and in consequence of the fall was unable to walk. Further questioned the mother said that her three children were playing in a bedroom with a miniature theatre, when one of the figures dropped from the stage and the child in rushing across the room to lift it tripped over her brother's foot and fell upon the floor. The brother raised her and set her on her feet, but she could not stand and the mother finding her in this condition carried her straight to my house. Although a bright healthy little girl she was very frightened and resisted examination, so I sent her home and saw her later. The objective appearances were marked distortion of the left hip and undue prominence of the gluteal region. The limb was adducted and rotated inwards, the thigh was bent slightly upon the abdomen, and the leg was flexed upon the thigh. The lower end of the femur crossed the lower third of the opposite thigh and the ball of the left great toe rested on the instep of the right foot. There were roughly about one and a half inches of shortening, though this observation was not verified by accurate measurement. The left trochanter lay well above Nélaton's line, and on rotating the limb the head of the femur could be felt considerably above the acetabular ridge. From these characteristics there was no doubt that I had to deal with a dislocation of the head of the femur on to the dorsum ilii.

Under chloroform, administered by one of my neighbours, reduction by manipulation was attempted but failed. Having, therefore, instructed the mother to fix the pelvis, the thigh was flexed at right angles to the abdomen and slow steady traction made in an upward direction. The limb was then adducted and rotated inwards, and the caput femoris slipped into the cavity of the acetabulum with a distinct click. The limb was put up in a Liston's long splint and secured by a spica bandage. At the end of four days the splint was removed and the patient was permitted to lie on a sofa until the eighth day, when she was allowed to go about and it was found that her recovery was complete.

With regard to the relative frequency of dislocations of the hip in children I have no statistics to offer, but I find that Erichsen tells of two cases which he met with in his own practice. In one of these—a child aged one and a half years—the head of the femur was dislocated on to the os pubis; in the other there was dorsal dislocation in a boy six years old. Erichsen also mentions that Kirby had seen a case of dorsal dislocation in a child aged three years, and that Madge had observed a similar case in a child aged three and a half years, while Travers had a dislocation of the hip in a boy five years of age.

Liverpool.

A Mirror
OF
HOSPITAL PRACTICE,
BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv., Proœmium.

BRADFORD ROYAL INFIRMARY.

A CASE OF TRAUMATIC PERITONITIS FOLLOWED BY TWO SEPARATE ATTACKS OF INTESTINAL OBSTRUCTION; THREE CŒLIOTOMIES IN LESS THAN THREE WEEKS.

(Under the care of Dr. C. F. M. ALTHORP.)

IT is extremely probable, nay, almost certain, that when a blow on the abdomen (not causing any perforating wound) is followed by peritonitis this is due to a rupture of some hollow viscus and the escape of some of the contents. If the aperture is large the peritonitis so produced will rapidly end in death unless by a cœliotomy the rupture can be dealt with surgically. When, however, the tear in the hollow viscus is exceedingly minute there may be only a very little extravasation and the peritonitis which is induced may be localised. The severity of the peritonitis depends chiefly on the micro-organisms which invade the peritoneum; if these be numerous and virulent the prognosis is very grave, but if few in number and comparatively harmless in character the resulting peritonitis will be less acute and serous in character. In the following case there can be little doubt that some minute rupture occurred, although it was not possible to discover the aperture at the operation; but the bacterial invasion of the peritoneum was not severe. The history, signs, and symptoms amply justified an operation, though it is not improbable that recovery would have ensued without any operation. For the notes of the case we are indebted to Mr. J. Phillips.

A boy, aged 11 years, was admitted into the Bradford Royal Infirmary on May 14th, 1901, with abdominal symptoms. While going home from school the previous afternoon he fell and struck his abdomen against a gate-post. He walked home (a few yards) and then was put to bed as he was suffering from abdominal pain and was "blue and cold." He was sick several times and next morning a medical man saw him and sent him into the infirmary. He was in perfect health before the accident. On admission the abdomen was slightly distended but fixed and board-like. Nothing was made out on palpation. There was dulness over the whole breadth of the lowest three inches of the abdomen. Liver dulness was present. The tongue was furred; the temperature was 100° F. and the pulse was 100. A catheter was passed and normal urine was drawn off. A normal motion followed an enema. The boy did not look so ill as would have been expected had there been any serious visceral lesion, but as there were undoubted peritonitis and effusion it was decided to explore the abdomen. An incision was made above the pubes, when a large quantity of yellow flaky fluid escaped. The appendix, cæcum, bladder, &c., all appeared to be normal. A sponge passed up towards the liver brought away small pieces of

lymph, so another incision was made above the umbilicus through which the liver, gall-bladder, stomach, and duodenum were examined, but no lesion was found. The abdomen was mopped clean and the wounds were closed without drainage. The boy did well for ten days and then complained of abdominal pain. The bowels refused to act and he began to vomit. On the twelfth day the vomit became intestinal and the lower wound was laid open. A coil of gut was found adherent to the abdominal wall immediately to the right of the incision and also to a piece of omentum. Both adhesions were freed and as no other cause of obstruction was found on hasty exploration of the abdomen the wound was sutured. The symptoms were relieved at once, the bowels being moved within two hours of the operation, and again the boy did well until the seventeenth day, when symptoms of obstruction again appeared. The lower wound was again laid open and the same coil of gut was found adherent as before but much more densely. In freeing it it was a good deal injured and consequently some six inches of it were resected, a Murphy's button being used. The boy made an uninterrupted and complete recovery from this operation; the button was passed on the seventh day after operation and after a lengthy stay in bed to allow the scars to become firm he left the infirmary in good health on July 23rd. He was seen and looked fat and well on June 5th, 1902.

Remarks by Mr. PHILLIPS.—This case was one of acute and diffuse peritonitis apparently traumatic in origin in which no visceral lesion was found to explain the etiology. It is reported chiefly because I have not been able to find in the literature any case in which a patient has had to submit to three cœliotomies in less than three weeks and it says much for the patient's vital powers that he survived them all, including so severe an operation as enterectomy.

DUMFRIES AND GALLOWAY ROYAL INFIRMARY.

LIPOMA SIMULATING SUBCLAVIAN ANEURYSM.

(Under the care of Dr. W. D. GRIEVE.)

IN the case recorded below a lipoma in the posterior triangle of the neck rested on the subclavian artery and apparently exerted some pressure on the brachial plexus. In many respects this swelling resembled an aneurysm of the subclavian artery, for which it was mistaken. It is possible that this lipoma was an example of what Verneuil described as a "supraclavicular pseudo-lipoma," but apparently in the case recorded below the growth was situated beneath the deep fascia. For the notes of the case we are indebted to Dr. A. K. Lauddie, house surgeon.

A man, aged 45 years, was admitted into the Dumfries and Galloway Infirmary on Dec. 12th, 1899, suffering from supposed left subclavian aneurysm. He was treated in the medical wards of this hospital for aneurysm for 39 days and was discharged as relieved. On August 20th, 1901, however, he returned suffering from acute septic cellulitis of his left hand and was admitted into Dr. Grieve's wards (surgical). He died from septic infection on Sept. 11th, 1901, and post-mortem examination revealed the supposed aneurysm to be a lipoma closely connected with the brachial cords and resting on the third part of the left subclavian artery and slightly on the scalenus anticus muscle. The patient said that about 20 years ago he noticed a small swelling and this went on increasing in size till about two years ago, when it stopped growing. He also complained of pain shooting down his left arm which was much thinner and weaker than the right. His left arm also continually trembled while he was awake but the trembling stopped when he was asleep. There was no history of syphilis or alcoholism, but of hard manual labour. On physical examination a swelling of about the size of a small walnut was seen pulsating in his left posterior triangle, which became more prominent as he sat up. The swelling was tender to touch and was felt to have slight expansile pulsation. On percussion it was found to be dull and on auscultation of the heart a distinct rough systolic bruit was heard over the aortic area and faintly over the swelling. Sphygmographic tracings confirmed all these signs of subclavian aneurysm and yet the swelling proved to be a lipoma.

Medical Societies.

ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

Causation and Treatment of Thrombosis occurring in Connexion with Typhoid Fever.

A MEETING of this society was held on Nov. 25th, Mr. ALFRED WILLETT, the President, being in the chair.

Professor A. E. WRIGHT and Dr. H. H. G. KNAPP said that in the hope of learning something of the causes of the thrombosis which was met with in connexion with typhoid fever they had addressed themselves to the task of making a series of comparative observations on the blood in (*a*) typhoid fever patients in the acute stage of the disease, (*b*) convalescents from typhoid fever, and (*c*) in normal persons. In each case the coagulation time of the blood and its content in lime salts were measured. They then described the method of determining (1) the coagulation time of the blood; and (2) the amount of calcium salts present in the blood. The results of the blood examinations undertaken on normal men showed that there was as between different individuals as well as between the blood drawn off at different times from the same individual, a considerable difference, not only with respect to the blood-coagulation time, but also with respect to the content in lime salts. It was found that the coagulation time might, in the normal male adult, vary between three and a half minutes and 15 minutes, and the minimum strength of oxalate of ammonium solution required to avert coagulation from 1 in 800 (this being altogether exceptional) to 1 in 2000. Of importance in connexion with the method of measuring the content of the blood in lime salts was the fact that a blood which contained less calcium salts than a control blood was not always less coagulable; nor, again, was a blood which contained more lime salts necessarily more coagulable than the control. The content of a blood in lime salts as estimated by this method was, in fact, far from being an index of its coagulability. The results of the blood examinations undertaken upon typhoid fever patients during the acute stage of the disease showed a general diminution of blood coagulability. The results of the blood examinations undertaken upon convalescents from typhoid fever showed a marked increase of blood coagulability. In eight cases—all of which were examined both during the course of the pyrexia and after the return of the temperature to the normal—it was found that the coagulation time of the blood was, on the average, four and a half times shorter in the convalescent stage than during the course of the fever. In four of these cases blood coagulation was abnormally rapid, and in each of these femoral thrombosis had supervened, and these were the only cases among those examined in which it had occurred. The results of the calcium salt estimations showed that the average content of the blood in lime salts, as estimated by the oxalate method, was in the case of convalescents from typhoid fever about twice that of the normal blood. It had been pointed out by Wooldridge that there supervened upon the "positive phase" of increased coagulability which culminated in the thrombosis a "negative phase" of diminished or abolished blood coagulability. By reason of the supervention of this "negative phase," the thrombus, when once formed, did not receive any further accretions of fibrin from the circulating blood. Quite different were the circumstances when the blood maintained its original coagulability. Here, as soon as any blood flow was re-established past the clot, additional fibrin would be deposited and the thrombus would grow larger and firmer until at last it was converted into a solid plug of "white blood-clot," which definitely blocked the vessel. Probably in this way were sown the seeds of the permanent trouble so often seen after thrombosis in typhoid fever. Recognising the practical importance of the after-deposition of fibrin upon the thrombus the coagulation time of the patient's blood subsequently to the development of thrombosis was a matter of concern. It was proposed, therefore, to place an obstacle in the way of this deposition of fibrin by diminishing the patient's blood coagulability and with this view a decalcifying agent—citric acid—was administered. In seven patients in whom observations were made it was found that the

exhibition of citric acid was followed by a decalcification of the blood and a corresponding diminution of its coagulability. Turning, in conclusion, to the problem as to what was the cause of the thrombosis so frequently seen in connexion with typhoid fever and scrutinising the results of the blood examinations to see whether they shed any light upon this problem, it was obvious that the quantum of lime salts in the blood of the convalescents from typhoid fever examined was greatly in excess of that in the normal blood. This fact suggested that the increased coagulability during the convalescent stage might be dependent upon an excess of lime salts. Evidence pointing in the same direction was afforded by the circumstance that the blood coagulation times of the typhoid fever patients, after the content of their blood in lime salts had been brought within the limits of the normal, were longer than those of normal bloods, instead of being shorter, as might have been expected if the increased coagulability were dependent upon an increase in the albuminous elements which enter into the composition of the fibrin. The excess of lime salts which appeared to be present in the blood of the convalescent from typhoid fever was probably derived from the milk which, for the most part, constituted the exclusive dietary of the patient. Cow's milk contained 1 part in 600 of CaO as compared with 1 part in 800 contained in lime water. If the milk dietary of the typhoid fever patient was the key to the problem of the frequency of thrombosis in the period of convalescence, a clue had probably been obtained also to the resolution of certain other problems, in particular the problem of the frequently beneficial effect of a milk dietary on "serous hæmorrhage" from the kidney and the comparative rarity of thrombosis after acute fevers such as Malta fever, where a milk dietary was not imposed upon the patient. At the same time indications for the prophylaxis and after-treatment of thrombosis were obtained both when it occurred in connexion with typhoid fever and when it occurred in connexion with other diseases. The remedial measure which would seem to be indicated was the exhibition of citric acid. The same treatment, initiated as soon as the danger of intestinal hæmorrhage had been surmounted, would be appropriate for prophylaxis of thrombosis in typhoid fever. Or, as an alternative, with a view to restrict the intake of lime salts, a partial decalcification of the milk might be undertaken. The partial decalcification in question could be readily effected by adding to the milk from 0·25 to 0·5 per cent. of citrate of soda (from 20 to 40 grains per pint).

Dr. WILLIAM HUNTER congratulated Professor Wright and Dr. Knapp on the paper and said that he had been in the habit of using sodium citrate in febrile conditions with beneficial results and Professor Wright had now given a scientific explanation of the effect of the salt.

Dr. H. D. ROLLESTON asked if Professor Wright attributed the whole process of phlebitis to the excess of calcium salts in the blood, for such phlebitis was usually attributed to infection of the wall of the veins. He commented on the greater frequency of phlebitis or thrombosis after typhoid fever in South Africa than in England and stated that the late Dr. J. W. Washbourn considered that it was dependent on the tinned food with which the patients were fed. He himself, however, considered that it was due to a diminished resistance owing to continual "trekking." He suggested that the condition of the patient was one approaching scurvy.

Dr. G. NEWTON PITT said that in most cases of thrombosis micrococci were present in the neighbourhood of the vessel. These might be the primary cause of the thrombus or might be deposited there after thrombosis had occurred. He asked if the excess of calcium salts in the blood could be accounted for by the excess of milk taken. Thrombosis frequently occurred after influenza and he did not think that that could be attributed to the excess of milk taken, and, again, infants who took milk exclusively did not suffer from thrombosis.

Dr. C. OGLE asked if the treatment by citric acid might not be attended by some danger of embolism, owing to the clot becoming dislodged from the wall of the vessel.

Professor WRIGHT, in reply, said that he had made no cultivation from the thrombosed vessels. He did not consider that lime salts were the only factor in causing thrombosis but that, given a high percentage of lime in the blood, a slight increase of the albuminous substance or of the white corpuscles would determine a thrombosis. In typhoid fever, however, the excess of lime salts was the principal factor.

MEDICAL SOCIETY OF LONDON.

Hæmatemesis: its Pathology, Prognosis, and Medical Treatment.

A MEETING of this society was held on Nov. 24th, Mr. A. PEARCE GOULD, the President, being in the chair.

Dr. BERTRAND DAWSON opened a discussion on Hæmatemesis: its Pathology, Prognosis, and Medical Treatment. He did not wish to make a lengthy systematic reference to the etiology. The source of bleeding might be (*a*) focal, coming from one vessel; or (*b*) diffused, coming from several small vessels or capillaries. It was not possible to determine from either the quantity or quality of blood the source in any given case. In portal obstruction the source of hæmorrhage was in the majority of cases from the œsophageal veins. Out of 42 cases of hepatic cirrhosis in which the veins of the gullet had been specially examined 35 were found to be dilated, and in 16 of these latter the apertures in the veins had been actually seen. The cause of hæmorrhage in anæmic young women was then discussed. In such cases severe and even fatal bleeding might take place by oozing from quite superficial erosions of the mucous membrane. Such hæmorrhages might precede actual ulceration, though they favoured its production. The subject of "vicarious menstruation" was next discussed. There was a widespread tendency to bleeding during the menstrual period from various surfaces; and these cases of hæmatemesis, like post-operative hæmatemesis, were most probably due to nervous influences. Out of 200 collected cases of so-called vicarious menstruation the bleeding was from the stomach in 32 cases. Cases of "gastralgia with hæmatemesis" were then referred to and the occurrence of hæmatemesis without definite lesion being found after death or during operation was noted. Turning to the subject of treatment Dr. Dawson thought that the medical and surgical aspects should not be separated from one another. As regards the medical treatment, the diet should be carefully adjusted. It was only rarely necessary in emaciated cases to feed by enemata. A quarter of a grain of morphia should be administered hypodermically. Chloride of calcium to promote the coagulation of the blood should be given per rectum. The hypodermic injection of ergot was harmful in focal cases, and in all cases it increased peristalsis. Adrenalin was more useful. Normal saline solution not exceeding 400 cubic centimetres in amount should be injected hypodermically when the quantity of blood lost was very great. Medical treatment was successful in at least 90 per cent. of the cases of gastric hæmorrhage. In acute hæmatemesis operation should never, or very rarely, be performed, but after one severe hæmatemesis in cases where the symptoms pointed to gastric ulcer the indication for the operative treatment of the ulcer was much stronger.

Dr. I. BURNEY YEO referred to the great difficulty of determining the cause of the hæmatemesis in any given case and he quite agreed that there was generally a too great readiness to diagnose ulcer of the stomach. There was no doubt in his mind that certain cases of hæmatemesis were associated with the menstrual period though their precise pathology was obscure. Reference was made to a series of cases published by Professor W. Osler of "anæmia splenica" in which the spleen had been excised with manifestly good results. Dr. Yeo took exception to surgical statistics bearing on the fatality of hæmatemesis. For instance, Mr. C. W. Mansell Moullin had given the case-mortality of hæmatemesis treated medically as 6 or 7 per cent. These figures did not at all agree with his own experience or that of other physicians, which only amounted to 3 per cent. Surgeons' statistics were probably derived from hospital cases among the poor and destitute who were placed in very unfavourable circumstances for medical treatment. Turning to treatment there was a spontaneous tendency towards recovery in this as in all hæmorrhages. Local and general rest was important; opium, especially by mouth or rectum, favoured this and was beneficial. But he was not in favour of the hypodermic administration of morphia. Two other drugs were beneficial, perchloride of iron and nitrate of silver (from one-third to one-quarter of a grain) combined if necessary with opium. The latter was specially beneficial when simple erosion was suspected. Ergotin had been widely advocated. Gallic acid was a drug in which the late Sir William Jenner placed the greatest faith for the treatment of gastric or intestinal hæmorrhage. Adrenalin

was apt to raise the general arterial tension. The subcutaneous injection of a solution of gelatin had been attended with unfavourable results in some cases. There was often too great a desire to feed the patient who could do very well for several days without any food at all. Operation might be suitable for poorer patients.

Dr. W. HALE WHITE remarked that the greater number of cases of hæmatemesis met with at the bedside arose from ulcer, cirrhosis, or malignant disease. By far the greater number of cases diagnosed at the bedside as "simple ulcer" occurred in women, the proportion being five women to one man. 50 per cent. of these female cases were in the decade 20 to 30 years; but only 18 per cent. of the male cases were in this decade, 40 per cent. being in the decade 40 to 50 years. This suggested that some of the women were suffering from a different disease. He disputed the statement that the mortality of gastric ulcer was five times more frequent in women than in men. He believed that the actual occurrence of gastric ulcer was really commoner in men than in women. Reference was then made to cases in which no ulcer was found either at the operation or at the necropsy. Hæmorrhage had been severe and ulcer diagnosed during life. In women aged between 20 and 35 years three symptoms—anæmia, dyspepsia, and hæmatemesis—might be associated without any ulceration of the stomach.[1] Possibly these cases were really an exaggerated condition of chlorosis. Such cases were, he believed, much less fatal than true gastric ulcer and they were benefited by iron. For the treatment of hæmatemesis he believed in the administration of half a drachm of perchloride of iron with an equal quantity of glycerine.

Dr. H. D. ROLLESTON first referred to some unusual causes of hæmatemesis. In chronic splenic anæmia periodic attacks of hæmatemesis, often at many months' interval, occurred and were regarded by Professor Osler as mechanical and due to obstruction to the return of venous blood from the cardiac end of the stomach. As bearing on this a case of splenic anæmia of twelve years' standing was referred to. Hæmatemesis had occurred at intervals of five months during the last three years. Removal of the spleen was followed by fatal gastro-intestinal hæmorrhage, probably from ligature of the splenic vein producing great venous engorgement of the cardiac end of the stomach. In rare instances dysentery and typhoid fever gave rise to ulceration of the stomach and severe hæmatemesis. He had seen ulceration of the stomach in advanced chronic Bright's disease, and though it might have been a coincidence it was possible that the ulceration was secondary. As a very rare cause of hæmatemesis he referred to invasion of the stomach by a malignant growth arising elsewhere. He described a case in which a primary malignant growth of the left suprarenal capsule ulcerated freely into the cardiac end of the stomach and being very hæmorrhagic gave rise to fatal hæmatemesis. Most books mentioned the backward pressure of chronic heart disease as a cause of hæmatemesis, but Dr. Rolleston believed that it was very rarely responsible for hæmatemesis unless complicated by some further lesion. Minute erosions of the gastric mucous membrane might occur in general hæmic infections or intoxications, and as a rare event hæmatemesis occurred in pneumonia. A special form of minute erosion had been described by the late Dr Charles Murchison, by Professor Dieulafoy[2] (exulceratio simplex), and by Dr. J. Lindsay Stevens (pore-like erosion of the gastric arteries).[3] It occurred in young women, gave rise to profuse and repeated hæmorrhages, and might easily prove fatal. This was doubtless the same condition as that referred to by Dr. Hale White.[4] It differed clinically from gastric ulcer in not being accompanied by any tenderness on pressure. Its presence after death was probably often overlooked, but recognition was facilitated by leaving the stomach in Müller's fluid for some hours. It might occur at the cardiac end of the stomach. Its origin was not, as some had suggested, an inflammation of small lymphoid follicles, since those structures were chiefly limited to the pyloric region. With regard to the occurrence of hæmatemesis in the course of cirrhosis it was probably less frequent than was ordinarily supposed. In 80 cases of cirrhosis which died directly from the effects of the disease at St. George's Hospital only 26, or 32·5 per cent., had had hæmatemesis and of these only four died from hæmatemesis. In giving rest to the stomach by rectal feeding it was important to carry it out thoroughly and to allow no water by the mouth. Water by setting up peristalsis might lead to hæmatemesis as in a case mentioned.

Dr. W. ESSEX WYNTER thought that there was a distinct class of cases in which no ulceration existed. In his experience 80 per cent. of the cases of hæmatemesis in young women recovered, while that occurring in later life was very much more fatal. He had, moreover, been struck by the rarity with which definite organic lesions were found in the stomach in the deadhouse, although gastric ulcer was so frequently diagnosed during life. He did not favour the rectal administration of food. Adrenalin and gelatin administered internally had a local effect. He had recently tried adrenalin by the mouth and hypodermically and the results had been very different, the former being local, while the latter produced general constriction of the arteries.

The discussion was then adjourned until Dec. 8th, when the subject will be reopened by a paper by Mr. A. W. MAYO ROBSON (Leeds) on the Surgical Treatment of Hæmatemesis.

[1] THE LANCET, June 29th, 1901, p. 1819.
[2] La Presse Médicale, Jan. 19th, 1898.
[3] Glasgow Medical Journal, January, 1899.
[4] THE LANCET, June 29th, 1901, p. 1819.

OPHTHALMOLOGICAL SOCIETY.

Avulsion of the Eyeball by Midwifery Forceps.—Paralysis of the Upward Movements of the Eyeballs.—Nyctalopia (Retinitis Pigmentosa) in Five Generations.—Necessity for using Colour Names in Colour blind Tests.—Exhibition of Cases and Specimens.

AN ordinary meeting of this society was held on Nov. 14th, Mr. A. STANFORD MORTON, Vice-President, being in the chair.

Mr. SIMEON SNELL (Sheffield) related a case of Avulsion of the Eyeball by Midwifery Forceps. The child was admitted on the day following birth into the Sheffield Royal Infirmary (in June, 1902) with the left eyeball lying on the cheek, being connected with the orbital tissues by little more than bands of conjunctiva. There was a mark of the forceps above the left eyebrow. The particulars supplied by the medical man in attendance showed that the mother was a small woman with a very contracted pelvis. The pelvis was so small that it was thought it would be necessary to use the perforator, but ultimately the child was extracted by the aid of forceps and the eyeball was found to be in the condition stated. The globe was removed by severing the slight bands that held it to the orbital tissues. The optic nerve was found to have been torn through, leaving fully an inch attached to the eyeball.

Mr. SNELL also related a case of Paralysis of the Upward Movements of the Eyeballs. The patient was a man, aged 50 years. After being at work all day he returned home at 5 P.M., when he had his tea and fell asleep and remembered nothing until four o'clock the next morning. His wife called him three times during the night but he recollected nothing of it. When he awoke he felt very giddy and had difficulty in standing. He again went to sleep and remained so until 8 A.M. He then awoke with severe pain in the forehead and diplopia. When seen by Mr. Snell three days later (July 25th) the movements of the eyes were normal in all directions except upwards, but either together or separately the man was unable to raise them upwards. On attempting to look up the upper lids made the normal elevation but the eyeballs did not move and a rim of sclerotic was left visible above the cornea. The attempt also brought about contraction of the occipito-frontalis muscle and wrinkling of the forehead. Vision in each eye was $\frac{6}{9}$. At eight feet distance from an electric lamp and on the same level he saw two lamps, one being about an inch above the other. There was absence of other nerve conditions. The diplopia disappeared two days later and at the end of August he was able to move the eyes upwards, either singly or together, perfectly.—Dr. W. ALDREN TURNER regarded the case as due to a vascular lesion from its suddenness. Limitation to these particular movements pointed to the corpora quadrigemina being affected and recovery to the escape of the subjacent grey matter.—Mr. R. MARCUS GUNN drew attention to the fact that although some patients were quite incapable of looking upwards when told to do so, yet if they were asked to follow an object gradually moved upwards they could do so quite well. He asked if this had been tried in Mr. Snell's case.—Mr. SNELL, in reply, said that the patient was utterly unable to follow with the eyeball any moving object upwards.

Mr. SNELL also described Nyctalopia (Retinitis Pigmentosa) occurring in Five Generations. He mentioned the case of a man, aged 46 years, who since childhood had found great difficulty in going about in the dark. Though gradually failing, the sight had become so bad during the last two and a half years that he was incapable of following his occupation. He was now incapable of finding his way about in the dark; the right vision = $\frac{2}{60}$ and the left $\frac{1}{60}$. The ophthalmoscope disclosed a typical picture of retinitis pigmentosa. The peculiar interest centred in the fact that he was a member of a family in which large numbers were similarly affected with night blindness. The first who was known to be affected was his great-grandfather. He left an only daughter who was afflicted and from her had descended numerous instances of nyctalopia. Beginning with the great-grandfather there had been a total of 69 individuals and of this number there was good evidence that 28 were affected with night blindness. The patient's father had 11 children—five were afflicted and six were normal-sighted; the patient himself was the father of 11 children, seven being affected—namely, five boys and two girls: the rest escaped. There was no evidence of consanguinity in the family. Nyctalopia descended in all instances without a break, no generation was skipped over, and it ran equally through the male and female lines. There was no instance of a normal-sighted son or daughter by the grandmother having transmitted the malady to their children or grandchildren. 15 of the afflicted ones were females, 10 were males, and of three the sex was not ascertained. Night-blindness showed itself in early childhood. At about the age of 40 years, some more and some less, the affected ones had become practically blind.

Dr. F. W. EDRIDGE-GREEN read a paper on the Necessity for the Use of Colour Names in a Test for Colour-Blindness in which he said that the first requirements of a test for colour-blindness were that colour names should be used and the person to be examined should employ and understand the use of the colour names—red, yellow, green, and blue. No test which ignored colour names could be efficient. He had predicted that if colour names were ignored in the Board of Trade tests normal-sighted persons would be rejected and this prediction was fulfilled. Over 38 per cent. one year and more than 42 per cent. another year were found to be normal-sighted and to have been wrongly rejected. An engine-driver or sailor had to name a coloured light when he saw it, not to match it. He had to say to himself, "This is a red light, therefore there is danger," and this was practically the same as if he had made the observation out loud. Even the method of matching colours should, in order to be efficient, be one of mentally naming them. In his own classification test he used coloured materials of different kinds, as similarity, other than defined by the word "colour," was the great source of error in a test of this kind. The colour-blind might be divided into two distinct classes, which were independent of each other but which might be associated. The first class included those who were not able to see certain rays of the spectrum; their spectrum was shortened at one or both ends. If a man had shortening of the red end of the spectrum he would not be able to see a red light at a distance though he might be able to pick out all the green wools in the classification test. A man of this kind when shown the red light of Dr. Edridge-Green's lantern test declared that there was no light visible, at once demonstrating his incapacity. The second class of the colour-blind included those who made mistakes not because they could not perceive a certain colour but because they were not able to recognise the difference between the colours which was evident to normal-sighted persons. Both these classes were represented by analogous conditions in the perception of sounds. The first class of the colour-blind was represented by those who were unable to hear very high or very low notes—that was to say, those notes were non-existent to them. The second class was represented by those who possessed what was commonly called a defective musical ear. Normal-sighted persons saw six definite colours (points of difference) in the spectrum. The second class of colour-blind persons saw five, four, three, two colours, or only one colour, according to the degree of the defect; and they confused the colours apparent to the normal-sighted which were included in one of their own. If the normal-sighted be designated "hexachromic" those who saw five colours might be called "pentachromic," those who saw four colours "tetrachromic," those who saw three colours "trichromic," those who saw two colours "dichromic," and the totally colour-blind "monochromic." The degree of the defect would be recognised by the names given to different colours. The pentachromic would miscall orange. The tetrachromic might in addition make mistakes with regard to blue. It was not necessary to reject either of these two varieties, because he had never succeeded in making them confuse the colours red, yellow, green, and violet. The trichromic were always in difficulty over yellow and miscalled it red, green, or red-green, and for practical purposes must be excluded as colour-blind. The dichromic confused red, orange, yellow, and yellow-green on the one hand and blue-green, blue, and violet on the other.—Mr. C. DEVEREUX MARSHALL agreed with Dr. Edridge-Green. A "look-out" or an engine-driver had not to match colours at all, but simply to recognise as quickly as possible a white or coloured light. It was most striking to find a person who could pass Holmgren's test with ease saying that a dull red light which was quite visible to a normal-sighted person did not exist at all and that the room was quite dark. Those persons with a shortened red vision were of the utmost danger when placed in responsible positions either at sea or on the railway. He wondered that the Board of Trade did not recognise this.—Mr. W. T. HOLMES SPICER asked whether Dr. Edridge-Green would not demonstrate his cases and his method of testing, for at the present time most people used Holmgren's test because they knew of nothing better.—Dr. EDRIDGE-GREEN said that he would take an early opportunity of doing this.

The following cases and card specimens were shown:—

Mr. JESSOP: A case of Sympathetic Ophthalmitis with Cysts of the Iris.

Mr. G. W. ROLL: A case of Congenital Ophthalmoplegia.

Mr. N. BISHOP HARMAN: Two cases of Fissura Facialis.

Mr. MACCALLAN: A case of Lymphangiectasis of the Conjunctiva with Microscopic Sections.

Mr. GRIMSDALE: Paralysis of both External Recti.

Mr. ARNOLD LAWSON: Paralysis of the Sympathetic.

MEDICO-PSYCHOLOGICAL ASSOCIATION.

Lunacy and the Law.

AT the meeting of this society on Nov. 20th an address was delivered by Sir WILLIAM R. GOWERS upon Lunacy and the LAW. This address appeared in full in THE LANCET of Nov. 22nd, p. 1369. The following is a report of the discussion which followed the reading of the address.

The PRESIDENT (Dr. J. WIGLESWORTH), in conveying the thanks of the meeting to Sir William Gowers for his paper, said that it was mutually advantageous that hospital physicians should at times give the association the benefit of their experience on subjects specially concerning alienists whose tendency was towards a too narrow specialisation. There could be no question about the social importance of the subject and he trusted that the discussion would result in some action being taken. There were a number of "border-line" cases which were habitually certified but which he thought should be treated without certification. Asylum men did not usually see many such cases, but he had had several.

Sir WILLIAM S. CHURCH, Bart., could not claim to have had much personal experience of the subject under discussion, but he agreed that it was one of very great importance. Physicians had at all times recognised the great hesitation with which "border-land" cases had been dealt with and he had personally entertained the most wholesome dread of being in any way mixed up with them. Sir William Gowers had made out a very strong case for some alteration in the law concerning certification. The strongest argument brought forward was based on the law of Scotland, which appeared to have worked well. He regarded the six months rule in Scotland as a very wise one, because he believed that a person who did not recover his mental balance in six months would probably not do so for a very long time. He strongly urged that there should be no breaking of the law, whatever one's views upon that law might be, so that no one could point a finger at a member of the profession and charge him with having broken the law. There should be a resistance to the temptation to make oneself a martyr in the matter. He believed that at the present time the Lord Chancellor had a

Bill for amending the Lunacy Act, and it was not due to laxity on his lordship's part that that Bill had not become law, because it had twice passed the House of Lords. He thought that the present was a very good time for pressing the matter forward. He could not help thinking that a proposal which so strongly appealed to common sense as the present one would receive the most favourable consideration.

Dr. GEORGE H. SAVAGE said that the subject had his most sincere sympathy and he had felt for many years that something of the kind was needed. Referring to the case of Dr. Broadhurst, who was prosecuted and afterwards committed suicide, he maintained that the fact of a man being of unsound mind did not necessarily imply that he was a person who ought to be detained. After all, insanity was a negation and the degrees of that negation varied immensely. He felt that the proposed alteration in the law would be good for the patient in every way. A patient who was seen and recognised to be of unsound mind and who could not be properly treated at home should be notified, and there would be no greater difficulty in his case than in a case of small-pox or measles. Of course, such people and such places should be visited; that would be essential. The proposed alteration would not only be more just all round but would result in an increase in the proper inmates for asylums. Dealing with Sir William Church's remark, he said that alienists felt that there were cases in which it was necessary to be above the law.

Sir WILLIAM H. BROADBENT, Bart., F.R.S., thought that the keynote of the matter was that no man should be certified as insane unless it were either for his own advantage or in the interests or safety of the public. To be certified was practically a sentence of imprisonment much more severe than the worst criminals were exposed to, owing to the mental suffering involved, and if such a person got better he felt that his self-respect was wounded for ever. He had no idea that the law was so strict as it turned out to be. No doubt he had been guilty of breaking the law in a few cases and, in spite of what Sir William Church had said, he would break it again rather than send to an asylum a person whom it was not necessary to send there on the pleas which he had laid down.

Mr. H. F. HAYES NEWINGTON said that not only would the association flatter Sir William Gowers by following his advice, but it had already flattered him by anticipating it to some extent. He detailed the steps which had been taken by the parliamentary committee of that association and of the British Medical Association to bring about an alteration, their suggestions going even further than those of Sir William Gowers.

Dr. DAVID YELLOWLEES (Glasgow) gave his experiences in connexion with the working of the Lunacy Law in Scotland, and spoke highly of the six months certificate in that country for incipient cases.

Sir JOHN BATTY TUKE spoke earnestly in support of the proposed alteration and strongly seconded the remarks of Dr. Yellowlees.

Dr. D. FERRIER expressed his thorough agreement with all that Sir William Gowers had said and remarked there was no doubt that unsoundness of mind was considered to be a stigma, though it ought not to be so.

Dr. G. F. BLANDFORD reminded the meeting of the long-continued efforts of the society to bring about reform and the wearying delays incidental to legislative enactments.

Dr. H. RAYNER pointed out the importance of trying to get the right kind of people to take charge of the patients, which was not by any means a satisfactory feature of the subject at present.

Dr. C. A. MERCIER made a very effective speech in favour of reform and tilted humorously at the inconsistencies of the law on the subject.

Dr. ERNEST W. WHITE appealed for enactments requiring that only thoroughly trained people should have charge of patients. He remarked on the objectionable habits acquired by well-to-do patients, especially females, compared with those of the poorer and agricultural classes.

Dr. URQUHART reminded his hearers that the association had long called upon the physicians of the country to come to the aid of the alienist in the matter. He supported the observations of Dr. White.

Dr. ROBERT JONES proposed a hearty vote of thanks to Sir William Gowers for his address, which was carried by acclamation, and it was resolved to refer the matter to the parliamentary committee of the association.

Sir WILLIAM GOWERS briefly replied.

ROYAL ACADEMY OF MEDICINE IN IRELAND.

PATHOLOGICAL SECTION.

Dislocation of Shoulder.—Exhibition of Specimens.

A MEETING of this section was held on Nov. 7th, Professor E. H. BENNETT being in the chair.

Professor BENNETT exhibited specimens of Dislocation of the Shoulder and read a note on some cases in which Kocher's method of reduction of dislocated shoulder had failed. The specimens were a dissection of an old unreduced dislocation, which in all particulars conformed with Malgaigne's definition of subclavicular dislocation, and a cast of the limb exhibiting the characteristic features of the injury. The cases recorded were two from the living—one seen on the day of the injury and the second five weeks after. In the first an attempt at reduction, made by very competent hands, had already failed. The second was five weeks unreduced and had been five times submitted to attempts at reduction under anæsthesia. The injuries conformed in their features with the subclavicular dislocation of Malgaigne; in both of them Kocher's manipulation and the method by the heel in the axilla failed. The reduction was in both obtained by traction with rotation on the arm abducted downwards and outwards. Professor Bennett discussed the influence on the reduction of the character and position of the lacerations of the capsule. Kocher's method was useful because it was applicable particularly to the intracoracoid dislocation, which was the most common, or its immediate modifications. He had never met with a case which resisted Kocher's method except the two he had described.

Dr. H. C. EARL showed a case of Adenoma occurring in a Liver which, he said, presented most marked cyanotic atrophy. The liver contained 15 or 20 small round masses of a green colour, the largest of them being about half an inch in diameter. The cells in them bore a close resemblance to liver cells. They were divided into lobules and were surrounded by a fibrous capsule. The cells were arranged after the manner of normal liver cells, but with less regularity. Coagulated bile was present and there was a great deal of granular bile pigment in the liver. Sections of some of the smaller tumours showed more marked resemblance to normal liver structure. These tumours were not malignant and appeared to be one of the forms of adenoma. He presumed that cyanotic atrophy of the liver had nothing to do with these.—Dr. W. R. DAWSON, Professor O'SULLIVAN, and Professor A. H. WHITE made some remarks.

Dr. EARL also described a specimen of Cavernoma of the Liver which he said was a well-recognised and not uncommon condition occurring both in human beings and in the lower animals. Cavernomata were sometimes single, but generally, as in this case, multiple, and usually occurred in elderly people, though sometimes they were seen at a very early age, even in infants a few weeks old. They appeared of a dark purple colour on account of the contained blood and were of various sizes. They might be as large as an apple. They were not raised above the surface of the organ when they abutted on it and they were also found in its substance. Their structure resembled that of the cavernous tissue of the penis. Simple muscular tissue was said to occur in their walls. In some cases liver cells had been found in their walls. The larger masses were surrounded by a fibrous capsule, the smaller ones merged in the liver substance without any capsule. There was considerable doubt as to their pathogenesis. They had been considered tumours but they never caused any enlargement of the liver. They were merely substitutions of the liver tissue and were generally believed to be due to dilatation of lobular cavities with subsequent pressure atrophy.

Professor J. A. SCOTT exhibited an Epithelial Tumour of the Gall-bladder removed by operation by Mr. F. T. Heuston.

Mr. ARTHUR H. BENSON and Dr. EARL showed a case of Conglobate Tuberculous Tumour of the Choroid in a girl, aged 14 years. The growth, which only dated from March 27th, 1902, occupied almost the whole interior of the globe, the vitreous being pushed to one side and the lens remaining transparent throughout. This variety of tuberculosis of the choroid was stated to be a very rare affection, whilst the miliary variety was comparatively common. It

was a remarkable circumstance that there was no tuberculous family history, nor was there any other focus of tuberculous disease to be found in any other part of the patient. The sections, which were made by Dr. Earl, showed that caseation had taken place in the tumour and giant cells were also visible. The growth was adherent to the sclerotic in one place and the retina was wholly detached. The other eye was normal and the child, though naturally frail, seemed to be in excellent health.—Mr. J. B. STORY said the case reminded him of a specimen which he had exhibited some two years ago. It was one of a solitary tuberculous tumour in a woman, aged 18 or 20 years. It was in the optic papilla and simulated the choked disc of intracranial disease. The growth in this case was retinal rather than choroidal.

FORFARSHIRE MEDICAL ASSOCIATION.—A meeting of this society was held at University College, Dundee, on Nov. 6th, Dr. A. J. Duncan being in the chair.—Professor D. MacEwan showed a girl after treatment for Neglected Talipes Equino varus. The treatment adopted was that of removing the interior of the bones which maintained the parts in the deformed position, the treatment introduced by Professor Ogston. Professor MacEwan showed by lantern-slide the patient's condition before treatment and pointed out the advantages of this operation in cases of inveterate club feet.—Dr. A. P. Low showed a child who had developed a Hæmatoma of the Right Orbit following delivery by forceps. He stated that at birth there was no bruising observed, but in five hours some swelling and discolouration were noticed in the right eyelids. The effusion continued for three days and forced the eyeball out of the orbit. Superficial sloughing of the cornea and perforation occurred. In three months the extravasation was completely absorbed and the eyeball had returned to its normal position, but the cornea was replaced by a dense cicatrix. The child was quite healthy. The mother was 44 years of age, multiparous, and had had no other complicated labours.—Professor MacEwan showed photographs on the screen of a case, a female child, with Congenital Absence of the Perineum before and after treatment. The development had stopped at the formation of a septum between the rectum and the vagina. He had endeavoured to form a perineum by flaps from the buttocks with some success. Professor MacEwan referred to the extreme rarity of such a case.—Professor J. A. C. Kynoch showed an Œdematous Fibroid Tumour of the Uterus successfully removed by abdominal myomectomy from a woman aged 30 years. The tumour had increased from the size of an orange to that of a fœtal head in three months as the result of œdematous degeneration.—Mr. D. M. Greig showed the Right Half of the Lower Jaw removed from a woman, aged 31 years, for Myeloid Sarcoma in August of this year. A swelling appeared in January, 1901, at the angle of the right lower jaw, but disappeared after the extraction of two teeth. It recurred in January of this year. In May, when Mr. Greig first saw the patient, the swelling was hard and tender to touch and there were two slightly enlarged glands below it. The patient's tuberculous appearance and unsatisfactory family history seemed to favour the diagnosis of tuberculous ostitis. Under chloroform the diseased tissue was scraped and part was removed for examination by Professor L. R. Sutherland who reported that the swelling was a myeloid sarcoma. The specimen shown was removed in August and the patient made an excellent recovery, due partly, Mr. Greig considered, to the use of anti-streptococcic serum. In connexion with this he stated that the free use of the serum produced effusion into the sheaths of the extensor tendons. One thing noticeable in this case was the varying size of the tumour, which Mr. Greig referred to, and also the impossibility of having an interdental plate fixed in this and similar cases where half of the lower jaw is removed.—Dr. G. Halley then gave notes of a case of Excision of Portion of the Left Half of the Lower Jaw for Myeloid Sarcoma in a girl, aged 12 years. The patient was first operated upon by Mr. Greig in August, 1899, for swelling in the left lower jaw of six weeks' duration; at that time—after a portion of the tumour had been removed, examined and found to be of the nature of myeloid sarcoma—part of the bone of the jaw was removed and the actual cautery applied. A swelling reappeared six months later and was of about the size of a pea and was excised deeply and a tooth extracted. Six months later than this, in October, 1900, a swelling reappeared at the angle of the left lower maxilla. The proximity of the tumour to the angle of the jaw, the reappearance of the swelling despite previous treatment, the nature of the tumour, and necessary interference with the muscles of the jaw if recurrence were to be prevented, decided Dr. Halley (under whose care the case had now come, in the absence of Mr. Greig in South Africa) to perform complete excision of the left lower maxilla posteriorly. The operation was performed on Nov. 5th and the bone was sawn through at the side of the left canine tooth, thus allowing an interdental plate to be inserted later. The plate was inserted a week later and the patient was sent home three weeks after the operation, retained the plate for about seven weeks, and was then able to masticate with the right side of her jaw. Dr. Halley stated that he had lost sight of her and could not speak of her present condition. Mr. Greig, who had seen her since Dr. Halley had, said that she was quite well and also considered that the interdental plate succeeded in Dr. Halley's case because he was able to leave so much of the lower jaw.—Dr. W. E. Foggie read notes of a case of Meralgia Paræsthetica which he had met with in a woman aged 50 years. There were present over the antero-external region of the right thigh the paræsthesia, the anæsthesia, and the recurring pains which together made up the cardinal features of the fully-developed condition; since its recognition seven years ago a considerable number of cases had been reported. The medical treatment had not been very satisfactory except in the milder cases but since the adoption of resection of the offending nerve excellent results had been obtained. Dr. J. Mackie Whyte said that he had seen a number of cases allied to Dr. Foggie's but without the paroxysmal pains.

SOCIETY OF MEDICAL OFFICERS OF HEALTH.—A meeting of this society was held on Nov. 14th, Dr. J. Spottiswoode Cameron, the President, being in the chair.—Dr. H. Meredith Richards (medical officer of health and medical officer to the School Board of Croydon) read a paper on the Sanitary Control of Schools, with special Reference to the Education Bill. After alluding to the evidence adduced by the late Sir R. Thorne and the investigations of Mr. Shirley F. Murphy as to the dissemination of infectious diseases through the aggregation of children in public elementary schools, he pointed to the fact that nearly 6,000,000 children, forming a sixth of the entire population of England and Wales, passed a large part of their time in these buildings, for the sanitary conditions of which the State was directly or indirectly responsible. School boards and managers needed better guidance as to the size, ventilation, lighting, and sanitary arrangements of new buildings and the adaptation of old ones than that of the district surveyor who, as a rule, would pass any plans involving no infringement of the local by-laws. Overcrowding, furniture in relation to posture, books, &c., required skilled medical control, and teachers should be instructed in the laws of health and the early recognition of infectious diseases which they should report at once to the medical officer of health, meanwhile excluding the suspects from school. A few schools had appointed medical officers, but their jurisdiction did not extend to the voluntary schools. The medical officer of health, save where, as at Croydon, he had been constituted also medical officer to the school board, could only order the closure of the schools or the exclusion of individual scholars likely to be the means of spreading infection, but he had no official status *quâ* schools and no right of entry on school premises. In some towns the medical officer of health had with the best results organised a system of voluntary notification by the teachers of all cases of infectious disease whether legally notifiable or not, but the full benefit to the community would not be secured until such notification was made obligatory, and the medical officer of health was empowered to inspect school premises and to enforce his recommendations as to sanitary defects. In Scotland the necessity for such control had been recognised by the Local Government Board and the education department, which had issued joint circulars urging coöperation between the school boards and the sanitary authorities. In England the sanitary conditions of schools were provided for by Article 85 and the procedure as regards infectious diseases was prescribed in Articles 88 and 101 of the code. Subsection 1 of Article 85 was fairly satisfactory but this could not be said of subsections 2 and 3, for 80 cubic feet and eight square feet of floor space per head were grossly insufficient, and even the 120 cubic and 10 square

feet required in new buildings involved serious vitiation of the air and a propinquity of the children conducive to the spread of infection.—Dr. A. Newsholme then moved a series of proposals the tenour of which was that the Board of Education was asked to take measures for making the curriculum and surroundings of the children educated in State schools such that the children would gradually acquire some knowledge of the laws of health. After much discussion and amendment these motions were referred to the council with the view of bringing them before the Government in relation to the Bill now under the consideration of Parliament.—Dr. James Kerr (medical officer to the School Board of London) said that he would rather see the creation of a body of medical officers of schools than the imposition of the duties on the medical officers of health.

A meeting of the society was held on Nov. 21st, the President being in the chair, when the conclusions of a large and representative committee of the society in respect of the hygiene of bakehouses were considered at a full meeting and adopted. The proposals will be found embodied in a leading article upon the subject on p. 1472 of this issue of THE LANCET.

LARYNGOLOGICAL SOCIETY OF LONDON.—A meeting of this society was held on Nov. 7th, Mr. E. Cresswell Baber, the President, being in the chair.—Mr. P. de Santi showed a case of Tertiary Syphilis of the Larynx in a man, aged 26 years. He had treated the patient four years previously for secondary syphilis, which cleared up. The patient returned in October, 1902, with hoarseness, some difficulty of breathing, and slight pain.—The President showed a case of Extensive Fenestration of the Anterior Pillars of the Fauces in a boy, aged 13 years, and raised the question as to whether it was the result of ulceration or congenital in origin. Sir Felix Semon thought that these cases were congenital, otherwise he saw no reason why the condition should be so often absolutely symmetrical.—Mr. C. A. Parker showed a case of Double Abductor Paralysis with Swelling of the Arytenoids in a man, aged 38 years.—Mr. R. Lake showed a case of Removal of the Epiglottis for Tuberculous Disease in a man, aged 35 years. The operation was undertaken for the relief of dysphagia and had proved entirely successful.—Dr. Scanes Spicer showed an improvised Working Model to demonstrate the air channels and currents in the nasal cavities in normal and impeded respiration. The results of experiments with this apparatus confirmed those of Paulsen and Franke on the cadaver and Parker's on the living subject.—Sir Felix Semon showed a case of Obscure Lardaceous-looking Variable Infiltration of the Uvula, Soft Palate and Right Arytenoid in a woman. On examination the uvula and the adjacent parts of the soft palate were seen to be considerably infiltrated and were smooth to sight and touch and of a peculiar yellowish colour, resembling the appearance of a lardaceous kidney. The right arytenoid presented a similar appearance. There was some pitting on pressure about the external malleoli but the urine had been found to be perfectly normal. The patient had been treated with arsenic and iron and the local condition had somewhat improved. Sir Felix Semon had seen two similar cases but did not think the condition had ever been described. He was unable to make any suggestion as to their pathology.—Dr. Fitzgerald Powell showed a case of Cyst of the Epiglottis which practically caused no symptoms.—Mr. A. H. Burt showed a case of Epithelioma of the Tongue in a single woman, aged 24 years.—Dr. H. L. Lack showed a case of Complete Adhesion of the Soft Palate to the Posterior Wall of the Pharynx.—Mr. Parker showed a case of Persistent Epistaxis in a man, aged 42 years, which had been going on for 18 months and had completely disabled the patient from work.—Dr. J. Dundas Grant showed a case of Paralysis of the Abductors of the Vocal Cords and of the Palatal Muscles with slight Paresis of the Tongue in a man, aged 25 years, which he considered due in all probability to a lesion of one of the vagus nerves in or near the medulla oblongata.—Cases were also shown by Dr. J. Donelan, Mr. F. Hunter Tod, Dr. E. Furniss Potter, and Dr. H. Tilley.

WINDSOR AND DISTRICT MEDICAL SOCIETY.—The second meeting of the session of this society was held on Nov. 19th, Mr. W. B. Holderness, J.P., the President, being in the chair.—Microscopical preparations were exhibited showing (1) Ulcer of the Ileum from a case of Typhoid Fever; (2) a Swollen Peyer's Patch from a similar case; (3) the Typhoid Serum Reaction—Positive; (4) the Typhoid Serum Reaction—Negative; and (5) Bacillus Typhosus showing Flagella. These specimens were kindly lent by Dr. G. Leslie Eastes.—The President showed two patients presenting characteristic physical signs of Mitral Stenosis.—Dr. C. R. Elgood showed: (1) A case of Hemichorea in a boy, aged nine years; and (2) a case of Advanced Mitral Disease with both Systolic and Diastolic Murmurs.—Surgeon-Major Beevor, C.M.G., of the Scots Guards delivered an address entitled "The Etiology of Typhoid Fever in Armies from Experience in the Egyptian, Ashanti, Tirah, and South African Campaigns." He commenced by enumerating the sources through which typhoid fever could be communicated to soldiers; these were water, soil, flies, and fabrics. He next demonstrated from his own experience of campaigns that water was not the sole channel by which the organism gained access to man. Among the examples given in support of this statement the case of the Seaforth Highlanders in the South African campaign was cited. This regiment, which was placed under no restrictions as regards drinking impure water, enjoyed an enviable immunity from typhoid fever all through the war. The question as to whether the disease could ever arise *de novo* was considered and cases were cited that would seem to warrant an affirmative answer. The dirt and general insanitary conditions that inevitably arose whenever human beings were crowded together were strongly dwelt on, and in this connexion the possibility of flies being the medium through which the poison was conveyed was considered. The various experiments which had been made with infected flies were alluded to and the lecturer also mentioned some interesting experiments made with soldiers' clothes, showing that the bacillus typhosus could reside in khaki serge for a considerable period of time.—Captain F. S. Brereton, R.A.M.C., in relating his experiences of a severe outbreak of enteric fever which occurred at Harrismith in the course of the South African campaign, stated that it certainly did not appear that the water had anything to do with the spread of the disease. He agreed with Surgeon-Major Beevor that at present the evidence pointed to the fact that in all probability flies were the principal medium through which the disease was conveyed to man.—The proceedings terminated with a vote of thanks to Surgeon-Major Beevor for his interesting address.

ÆSCULAPIAN SOCIETY.—A meeting of this society was held on Nov. 21st, Dr. Thomas G. Stevens, the President, being in the chair.—Mr. W. Campbell M'Donnell showed a section of an Acute Carcinoma removed from the skin of the flank of a woman, aged 76 years.—Dr. Alexander Morison read a paper to open a discussion on Heart Disease in Childhood. He showed a specimen of a heart weighing 22 ounces from a child, aged 10 years, whose aortic and mitral valves were defective and in whom the pericardium was universally adherent to the heart and the muscle was much overgrown. He thought that congenital heart disease might be due to fœtal endocarditis but more probably to a mere arrest of development and he recalled the fact that a defect of the ventricular septum was sometimes present with a defect of the palate, these two arrests occurring at about the same fœtal time. Auscultatory evidence of heart disease might be wanting yet other signs be conclusive. The prognosis in the congenital state was proportional to the fulness of the development of the heart. He had seen a case of acquired endocarditis in an infant under the age of four weeks in which infection seemed to be through the umbilical cord. The development of valvular affections in the child might be very insidious and might only be detected for the first time some weeks after an indefinite ailment. Pericardial adhesion was a common condition in the child's attacks and probably did much to induce hypertrophy beyond that needed for compensation of the defect of the valve. So this sequence being proved he suggested that when proved the pericardium should be exposed and the adhesions when practicable should be broken down by the finger. Prognosis of the child's heart was most uncertain for relapses of rheumatism were frequent and so added further damage. Rest of body, said Dr. Morison, was the chief point in treatment and to insure that salicylates, opium, and ice should be used to relieve the pain.—A general discussion followed and Dr. Morison replied.

BRISTOL MEDICO-CHIRURGICAL SOCIETY.—The second meeting of the session of this society was held on Nov. 12th, Mr. G. Munro Smith being in the chair.—Dr. A. Ogilvy showed a patient with Congenital Subluxation of

both Crystalline Lenses.—Dr. C. Walker and Dr. A. B. Prowse spoke on the condition.—Mr. A. W. Prichard showed a Tumour of the Omentum to which were adherent some four feet of small intestine. The entire mass was removed, the divided ends of the intestine were united end to end with the aid of O'Hara's forceps, and the patient was doing well.—Dr. W. H. C. Newnham showed a Tumour of the Uterus removed by Intra-peritoneal Hysterectomy.—Mr. J. Paul Bush, C.M.G., Mr. Munro Smith, and Mr. W. Roger Williams spoke on the subject.—Mr. J. Taylor read an interesting paper on the Clinical Use of High-Frequency Currents, first of all defining what was meant by the term "high frequency." He described numerous cases that had been under his treatment and he showed and explained the various forms of the apparatus used.—Mr. Munro Smith, Dr. W. C. Swayne, Dr. W. K. Wills, Dr. G. Parker, Dr. J. O. Symes, Dr. P. King, Dr. P. Watson Williams, and Mr. Roger Williams commented on the paper.—Dr. Watson Williams read a paper on a case of Aneurysm of the Aorta. He showed the specimen and pointed out the difficulty there was in diagnosing the case from one of mediastinal tumour.—Dr. E. Markham Skerritt, Mr. Roger Williams, Dr. E. J. Cave, Dr. T. Fisher, and Dr. J. Michell Clarke discussed the case.—Dr. Clarke read a paper on a case of Banti's Disease, and Dr. Fisher, Dr. Parker, and Dr. Symes joined in the discussion.

PATHOLOGICAL SOCIETY OF MANCHESTER.—A meeting of this society was held at the Owens College on Nov. 12th, Dr. H. A. G. Brooke, the President, being in the chair.—Dr. J. Orr gave a lantern demonstration of the Changes in the Central Nervous System in Six Cases of Acute Insanity. He described the different stages of the degenerative process found in the nerve cells of the cortex, of the spinal cord, and of the posterior root ganglia. Special attention was drawn to the fact that the majority of the cells affected showed a central chromatolysis and the frequency with which the nucleus showed grave degenerative changes was also demonstrated. Slides showing the atrophic process affecting the myelin sheaths in the lateral and posterior columns were also exhibited.—Dr. H. Ashby showed a Lardaceous Liver with Syphilitic Gummata from a girl, aged eight years.—Dr. F. C. Moore described the Hepatic Lesions met with in cases of Arsenical Beer Poisoning.—Mr. J. Collier and Mr. A. H. Burgess showed a specimen of Intestinal Infarction resulting from Embolism of an Intestinal Branch of the Superior Mesenteric Artery. The symptoms during life were those of intestinal obstruction of a very acute character.—Mr. Burgess showed the following card specimens: (1) a Recurrent Malignant Growth of the Bladder; and (2) Large Fibromyoma of the Cervix Uteri expanding the Cervical Canal and removed by Panhysterectomy.

MANCHESTER CLINICAL SOCIETY.—A meeting of this society was held on Nov. 18th, Dr. T. A. Helme, the President, being in the chair.—Dr. G. H. Lancashire read a paper on the X-ray Treatment of Skin Diseases. After dealing with the history of the method and giving a brief account of its technique, the paper described more in detail its use for the treatment of some varieties of lupus vulgaris and rodent ulcer. Some statistics were read relating to the treatment of the latter affection at the Manchester and Salford Hospital for Skin Disease. Out of 25 cases of rodent ulcer treated during the last 12 months, eight had been discharged apparently cured, 13 were still progressing favourably under treatment, while two had been unsuccessful.—Mr. Milner, Mr. David Owen, Mr. J. Rust, Dr. C. Christopher Heywood, and Dr. Taylor joined in the discussion.—Dr. J. Price Williams reported a case of Puerperal Eclampsia in the Seventh Month of Pregnancy treated by hypodermic injections of morphia without induction of labour, labour occurring naturally six days after the cessation of the eclampsia. Commenting on the differences of opinion as to the best method of controlling the convulsions he suggested that the society might undertake a systematic inquiry as to the utility of morphia for that purpose.

TORQUAY MEDICAL SOCIETY.—A meeting of this society was held on Nov. 20th, Dr. F. D. Crowdy, the President, being in the chair.—Mr. G. J. Arnold showed a child whose External Popliteal Nerve he had Resected and Sutured, it having been divided in opening an abscess some time previously. He also showed a patient with an improved Form of Spinal Plaster Jacket which gave support to both the occiput and the chin.—Mr. P. H. Gardner showed a remarkable case of recovery from a self-inflicted service revolver Bullet Wound of the Skull. The bullet entered far back in the right frontal region, passed through the brain, comminuted the bone at a corresponding place on the opposite side and lodged under the skin. The bullet and the fragments of bone were removed and a depressed fracture of the vault of the frontal bone, reaching from one wound to the other, was elevated. Large quantities of brain matter escaped before and after the operation but eventually the man made a good recovery. The visual field of one eye, however, was much diminished.—The President read a paper on the Early Symptoms of Tabes Dorsalis, giving the clinical history of several interesting cases, including one showing the rare condition of rectal crises.

MANCHESTER MEDICAL SOCIETY.—A meeting of this society was held on Nov. 5th, Dr. Thomas Harris, the President, being in the chair.—Mr. W. Thorburn reported three cases of Pyloric Obstruction not due to malignant disease—viz.: (1) a case of horseshoe kidney; (2) a case of hydatid cyst located in the concavity of the duodenum; and (3) a case of adhesions external to the stomach but arising from old ulceration of that organ.—Professor R. B. Wild made a communication upon the Use of Mercury in the Treatment of Syphilis and the Best Methods for its Administration.—Dr. Milligan made some remarks upon Subcutaneous Injections of Paraffin in cases of Saddle-Nose and other Deformities. He showed photographs of patients who had been operated upon, and furnished some details regarding the methods of procedure, the difficulties, and the results which might be reasonably looked for.—Dr. Milligan also showed a New Form of Syringe which he had designed for injecting purposes—the main feature of the syringe being that it was incased in an oil jacket and was connected with a compressed-air apparatus.

EDINBURGH ROYAL MEDICAL SOCIETY.[1]—A meeting of this society was held on Nov 21st, Dr. Wilson being in the chair.—Dr. Prentice delivered his Presidential Address, the subject being the Rational System of Therapeutics. He gave a survey of the treatment of all ages, but he dealt chiefly with the therapeutics of the present day.—Dr. T. W. E. Ross read an account of a case in which there were marked gastro-intestinal disturbance and nervous symptoms, occurring in a man, aged 25 years, a miner. There was a long discussion as to the nature of the case, at least six different diagnoses being suggested.—Dr. Dods Brown described a case which he considered to be Hypertrophic Cirrhosis of the Liver without Ascites.

Reviews and Notices of Books.

A Text-book of Practical Therapeutics, with especial Reference to the Application of Remedial Measures to Disease and their Employment upon a Rational Basis. By HOBART AMORY HARE, M.D., B.Sc., Professor of Therapeutics and Materia Medica in Jefferson Medical College of Philadelphia; Physician to the Jefferson Medical College Hospital; one time Clinical Professor of Diseases of Children in the University of Pennsylvania; Laureate of the Royal Academy of Medicine in Belgium, of the Medical Society of London; Corresponding Fellow of the Sociedad Española de Hygiene of Madrid. Ninth edition, enlarged, thoroughly revised, and largely re-written. Illustrated with 105 Engravings and 4 Coloured Plates. London: Henry Kimpton. 1902. Pp. 857. Price 21s. net.

WHENEVER several editions of a work have been called for and published in the course of a few years it may be said to have passed out of the hands of the reviewer. Notwithstanding that this has become a somewhat trite, because stereotyped, expression, the fact remains that the present is the ninth edition of this text-book of practical therapeutics. The first edition was published towards the end of the year 1890 and the eighth only so short a time ago as April, 1900,

[1] In our report of the meeting of this society published in THE LANCET of Nov. 22nd, p. 1396, the name of the fourth President should have been *Dr. S. A. K. Wilson*, not Watson.

and this bare statement of fact may be safely left to tell its own tale. The success of a work proves that its merits are generally recognised, that it supplies a need, and that it is largely independent of anything that reviewers can say about it. But we are in the present instance entirely in accord with the general professional opinion. The author in the preface to this ninth edition tells us in effect that in preparing the first he had endeavoured to present scientific facts and practical therapeutic measures in such a manner as to render the former interesting and the latter rational, and that in the new edition, while keeping these objects in view, he has made such changes, revisions, and additions as to render the volume still more useful to the practitioner and student. Considering its scientific nature we may say that the subjects are dealt with in a rational and interesting way and that there is nothing heavy about the volume which has the merit, so essential to a text-book of this sort, of being well arranged and supplied with a good and copious index. To deal with it, however, a little more in detail we may say that the title-page correctly describes its nature and that the work really fulfils what it sets forth. It is divided into four parts. The first deals with general therapeutical considerations, the second with drugs, the third with remedial measures other than drugs and with the feeding of the sick, and the fourth part with diseases and remedies, tables of doses of medicines and indices. One of the strong points of the book is the way in which the physiological effects of drugs are dealt with and illustrated. If the reader will leisurely and thoughtfully turn over its leaves, pausing here and there to select some particular subject for perusal *in extenso*, he will not only see how much useful information it contains, but in the course of his scrutiny he will come upon a number of new, interesting, or suggestive points. The remarks on alcohol, amyl nitrite, antipyrin, arsenic, belladonna, caffea, and ergot, and the pages devoted to chloroform, ether, nitrous oxide, and other anæsthetics, antitoxin, cold and heat as remedies, the feeding of the sick, and other subjects might, for example, be cited by way of illustration, but we have said enough to show that, in our opinion, "Hare's Text-book of Practical Therapeutics" is a very useful and capital work and one well deserving the success and popularity which it has achieved.

Grundriss zum Studium der Geburtshülfe, in Achtundzwanzig Vorlesungen und Fünfhundert fünfundsiebenzig bildlichen Darstellungen. (*Principles of Midwifery in 28 Lectures and 575 Pictures.*) Von Dr. ERNST BUMM, Ord. Professor und Direktor der Universitäts-Frauenklinik in Halle. Wiesbaden: J. F. Bergmann. Glasgow: F. Bauermeister. 1902. Royal 8vo. Pp. 778. Price 14s.

IT is most refreshing to come across a book with so many illustrations as this work of Professor Bumm and yet to find hardly a single example of the old and hackneyed drawings so well known to the reviewer. The author has certainly recognised the many advantages of numerous and good illustrations. Apart from their value from the educational point of view they render a text-book of this kind, not only most interesting and valuable to the teacher, but also make it more fascinating to the student. There is a great deal, however, to be noted with approval in this series of lectures besides the illustrations. The lectures are written in a clear and dogmatic fashion and present all the most important and recent views upon obstetric medicine. After a short historical introductory chapter 12 lectures are devoted to the physiology of the subject, while the remaining 14 are concerned with the pathology. Obstetric operations are discussed in a short appendix. The section upon the Anatomy of the Placenta and Decidua is well up to date and contains a clear account of the relations of the decidua to the ovum and the early stages of the development of the placenta. In discussing the diagnosis of pregnancy stress is laid upon the importance of abdominal palpation and the method of practising this recommended by Leopold is described and figured. A very interesting drawing is the one given in the section devoted to a consideration of the ring of Bandl. It is that of a uterus which had to be plugged for post-partum hæmorrhage and which shows no less than three retraction rings above the level of the internal os. In discussing incarceration of the retroverted gravid uterus the differences that exist when the uterus is only retroverted and when it is also retroflexed are clearly pointed out and illustrated. In cases where the condition has existed for some little time and where the mucous membrane of the bladder is in danger of necrosis opening the bladder through the vagina and draining it are recommended as the best method of treatment. No mention is made of the replacement of an incarcerated uterus by elastic pressure or by the introduction of a ring pessary into the vagina. Professor Bumm does not describe the method recommended and practised by some obstetricians of performing laparotomy for such cases when the uterus cannot be replaced—an operation which we think can never be necessary or, indeed, justifiable. One of the most interesting and instructive series of illustrations is that upon page 333 and the following pages showing the changes that may occur in the position of a fibroid tumour of the uterus during the course of pregnancy. How an intra-pelvic tumour may gradually rise out of the pelvis and its place be taken by the fœtal head is exceedingly well demonstrated and the case shows how often a fibroid tumour which in the earlier months of pregnancy appears to be certain to call for operative interference may, if the case be left until full time, finally cause no obstruction to labour at all—a clinical fact which some practitioners are apt to lose sight of in these days of abdominal surgery. It is to be regretted that no history is given of the case figured upon page 350, that of an interstitial gestation in which the uterus and fœtal sac entire were removed through the vagina. The view that in the development of a hydatidiform mole the covering epithelium of the villus plays an important part is confirmed and Professor Bumm appears to regard the two layers of epithelium as ectodermal in origin and a deciduoma malignum as really a variety of epithelioma of the chorion. A specimen of deciduoma malignum apparently beginning after the menopause, recently shown by Dr. F. J. MacCann before the Obstetrical Society of London, is one of very great interest and should throw a new light upon this debated question. Like other writers the author recognises two distinct varieties of hydatidiform mole, the simple form and the destructive form tending to penetrate the uterine wall and even leading to perforation, the so-called mola destruens, deciduoma malignum being distinguished from the latter by its tendency to form a definite tumour and the liability to the formation of secondary deposits in different parts of the body. A very fine example of the mola destruens from the Halle clinic is figured on page 375. In the treatment of an inevitable abortion Professor Bumm is in favour of active interference and clearing out the uterus in all cases where this can be performed under proper aseptic conditions. In private practice, however, he recommends a more expectant treatment. The chapter on Contracted Pelves is especially good and is illustrated by a large series of very clear figures of deformed pelves, of the mechanism of labour in these cases, and of the changes in shape of the child's head that occur. As a last resource in the treatment of accidental hæmorrhage the author recommends vaginal Cæsarean section. He says nothing about the use of Champetier de Ribes's bag introduced into the uterus without rupture of the membranes, a method of some value. Professor Bumm agrees with Ahlfeld's objection to Hofmeier's

views as to the development of a placenta prævia. Ahlfeld contends that the development of the chorionic villi in relation to the decidua reflexa can only give rise to what he terms a "placenta prævia spuria," because the connexion between the decidua reflexa and decidua vera will be so slight that very little bleeding will follow the separation of such a placenta prævia. Low implantation of the ovum in the neighbourhood of the internal os is held by the author to be the true explanation of these cases. Like most German writers upon the subject Professor Bumm regards the attachment of the placenta to the cornua of the uterus near the internal openings of the Fallopian tubes as one of the causes of uterine inertia and post-partum hæmorrhage and this view is illustrated by several drawings of such a condition. It is surprising that this is a matter upon which the great majority of English text-book writers are entirely silent.

The appendix, which treats of the various obstetric operations, is rather short and sketchy. No doubt it contains as much as is usually included upon this subject in a systematic course of lectures and it is intended that the students shall learn the practical details in their class of practical midwifery. Great credit is due to the publishers for the very excellent manner in which the illustrations have been reproduced and the whole work is worthy of the high reputation of its author.

Diseases of the Intestines, their Special Pathology, Diagnosis, and Treatment. By JOHN C. HEMMETER, M.D., Philos.D., Professor in the Medical Department of the University of Maryland. In two vols. Vol. II. London: Rebman, Limited. 1902. Pp. 679. Price 25*s.* net.

IN our review of the first volume of this work we were enabled to speak in terms of high praise of Dr. Hemmeter's labours. Our opinion has been strengthened after a perusal of the second volume. The various morbid conditions are described and the symptomatology and treatment are ably considered. In the volume now before us those pathological conditions are dealt with which occupy the border-land between medicine and surgery. The first chapter, which occupies 86 pages, is devoted to an elaborate description of appendicitis. The account of the anatomical features is very full and includes the observations which have been made by investigators in all countries; the plates in connexion with this part of the subject are well worthy of attention. Dr. Hemmeter speaks in no undecided terms when discussing the indications for operation. He remarks: "It can no longer be said that the question of therapeutic management of suppurative appendicitis and perityphlitis is whether it shall be purely expectant, or non-operative, or surgical. The views of American clinicians ..... and American surgeons ..... are now practically unanimous that this is a surgical condition to be treated by operation only." He then proceeds to point out the conflicting views held by various surgeons as to the choice of the time when the operation shall be performed. He wisely concludes that no definite rules can be formulated concerning the exact time when an operation is necessary and when not. "This must be decided in each individual case." Dr. Hemmeter unhesitatingly decides, however, against exploratory puncture and attempts to aspirate pus in cases of appendicitis. The whole question of operative intervention is again discussed in a later chapter of the book. We do not think this plan of dividing a subject an advantageous one, for it not only leads to a great deal of repetition but also to some confusion of ideas.

Chapter II. is headed "Infectious Granulomata of the Intestine." Tuberculosis of the intestine is first considered. Dr. Hemmeter criticises in an able manner the announcement made by Professor Koch at the British Congress on Tuberculosis in 1901—namely, that the infection of human beings with bovine tuberculosis is but a very rare occurrence. Dr. Hemmeter points out that the stomach and intestines give evidence of a "miraculous immunity" from tuberculous infection, which is in part to be ascribed to the activity of digestive ferments, in part to the peristalsis, and in part to some natural provision for disinfection which is not yet fully understood. Even if Professor Koch's inference that the human subject cannot be infected with the bacillus of bovine tuberculosis, we should still feel justified in assuming that primary intestinal tuberculosis should be of more frequent occurrence. A reason must therefore be sought to account for the rarity of the condition; either the bacillus can pass through the intestinal structure without leaving any trace of its passage or there is some normal provision for disinfection. These points must be the objects of further research. The subjects of Syphilis of the Intestine and of Actinomycosis of the Intestine also receive attention.

Chapter III., entitled "Intestinal Occlusion," practically forms a monograph on the subject, occupying as it does nearly 250 pages. Dr. Hemmeter is very properly extremely precise in his nomenclature and prefaces this chapter on the intestinal stenoses, occlusions, strictures, constrictions, obturations, and intussusceptions with an exact definition of these terms. This is a good precaution which might well be adopted by other authors. We have not space to give a full criticism of this chapter; it contains a vast number of interesting facts and will well repay careful study. The remarks on the use of opiates will be found of value. Some authorities deprecate the use of opiates in intestinal occlusions, maintaining that by their action the character of and symptoms of the disease is obscured, that the patient objects to an operation because of the apparent improvement, and that consequently the physician may be deceived in his prognosis and may lose the most favourable opportunity for an operation. This claim Dr. Hemmeter considers to be not sufficiently logical to justify the physician in omitting the administration of opiates and quotes Professor Naunyn to the effect that "it is not the privilege of the physician to withhold the benefits of his science from his patient in order to coerce him to a more correct view." Most authorities rightly urge that it is the duty of the medical attendant to ameliorate, as far as possible, all the patient's sufferings.

The chapter entitled "The Clinical Aspect of Intestinal Surgery, Borderline between Medicine and Surgery," will also be found to afford interesting reading. A large number of pathological conditions of the intestine call for immediate surgical interference, such as perforating ulcers in any part of the intestinal tract and malignant neoplasms when there would seem to be a reasonable chance of their complete removal. In other cases, however, the question of operation is a difficult one, but its importance can hardly be overestimated. In some conditions there is more or less debateable territory between the domains of medicine and those of surgery, which, according to the prevailing indications in any particular case, may at one time be conceded to the physician, at another to the surgeon. It is to such problems that the author devotes the sixth chapter in this volume. His views we believe to be sound and unprejudiced. We have already referred to his remarks on the operative treatment of appendicitis. He now elaborates his opinions by a careful review of the indications in the various types of the disease and reiterates his contention that if pus be present surgical intervention should not only be undertaken as a last resort, but that operation should take place much more frequently than hitherto (we presume that he is speaking of the procedure that has been adopted in America) and after more precise and more clearly-defined indications than have up to the present been laid down. Amongst other subjects considered in this volume are intestinal atrophy, abnormalities of form and position of the intestine, diseases of the intestinal blood-vessels and the influence of intestinal